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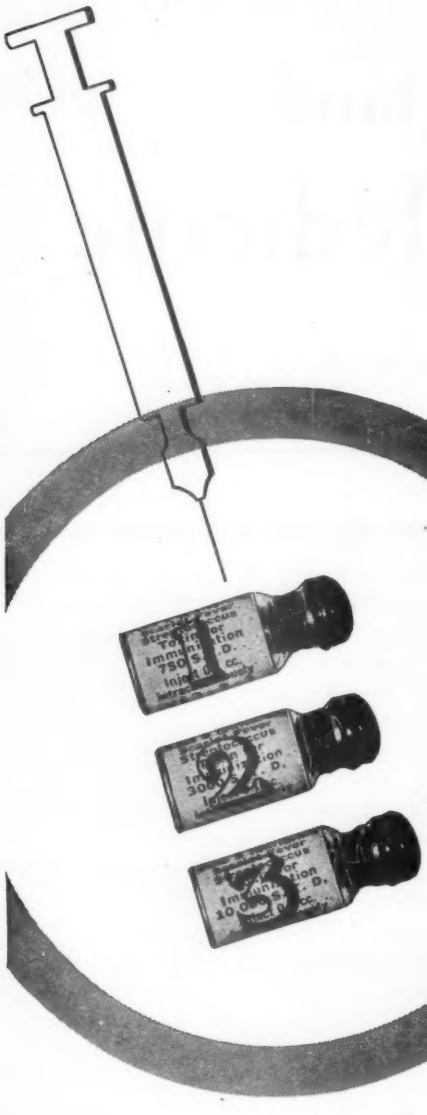

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Volume 234 JANUARY 10, 1946 Number 2

HODGKIN'S DISEASE*

VI. Clinical Diagnosis

HENRY JACKSON, JR., M.D.,† AND FREDERIC PARKER, JR., M.D.‡

BOSTON

IN THE last analysis, the diagnosis of any type of Hodgkin's disease must rest on a properly executed biopsy. It is true that in certain advanced cases of Hodgkin's granuloma with generalized lymphadenopathy, evidence of involvement of internal organs, fever, polymorphonuclear leukocytosis and anemia, the diagnosis can be made with some degree of assurance on clinical grounds alone, but even under these circumstances experience has taught us that we may be mistaken.

We therefore advise the removal of a lymph node whenever feasible. The node must be selected with care and with due reference to any possible surgical risks. In all cases, a fair-sized node should be removed, for the small satellites toward the periphery of an involved area not infrequently show merely nonspecific inflammatory changes, and it is not easy to persuade a patient to submit to a second operation for no better reason than that the first one was unsatisfactory. Similarly, a lymph node already subjected to irradiation should be avoided unless there is an express intention to study the results of therapy, for the effect of irradiation is largely cytolytic, and the characteristic cells on which the diagnosis rests may therefore have been destroyed. It is further of the utmost practical importance that the tissue be immediately preserved in a suitable fixative and subsequently be properly cut and stained. The histologic features of Hodgkin's granuloma cannot be identified in poorly prepared tissue, nor, for that matter, can those of any of the malignant lymphomas.

GENERAL CONSIDERATIONS

For convenience, the differential diagnosis of Hodgkin's disease may be grouped under three headings: patients with simple localized lymphadenopathy; those with generalized lymphadenopathy, with or without enlargement of the spleen; and those with constitutional symptoms of one sort or another, associated with little or no enlargement of the superficial nodes.

When confronted with simple lymphadenopathy apparently confined to a single region, one must consider various forms of lymphoma, — that is, giant-follicle lymphoma, reticulum-cell sarcoma, lymphosarcoma, Hodgkin's paragranuloma and Hodgkin's sarcoma, — as well as tuberculosis, carcinoma, chronic inflammation, infectious mononucleosis, syphilis, leukemia and certain other conditions.

Without biopsy neither Hodgkin's paragranuloma nor Hodgkin's sarcoma can be distinguished from Hodgkin's granuloma. In the first of these, however, there are few if any systemic symptoms. The peripheral blood reveals no abnormalities, and the patient's general health is at first unaffected. The course is notably long. Most patients with Hodgkin's sarcoma are middle-aged or elderly, the disease being extremely rare under the age of thirty. The patient's health is usually disturbed, often seriously so, even early in the course of the disease. Pain and infiltrative, destructive lesions of the viscera are frequent. Fever and leukocytosis seldom occur except in its terminal stages. The course is rapid, and spontaneous remissions are extremely rare.

Giant-follicle lymphoma is indistinguishable clinically from Hodgkin's granuloma in its early stages except by biopsy.

Reticulum-cell sarcoma occurs in the older age groups, with the significant exception of primary reticulum-cell sarcoma of bone. The involved nodes are usually hard, and sometimes even stony hard, and have a tendency to become fixed to the surrounding tissues. Pain is frequent and often severe. Fever and leukocytosis are rare. Localized reticulum-cell sarcoma is likelier to be mistaken for carcinoma than for Hodgkin's granuloma. This is particularly true when the disease affects the tonsils or the gastrointestinal tract.

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This is the sixth of a series of seven papers covering the various aspects of Hodgkin's disease.

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Lymphosarcoma, which is oftenest seen in childhood and in late adult life, is extremely rare between the ages of twenty and thirty years, a period of life during which Hodgkin's granuloma is frequent. The involved nodes that are the peripheral expression of lymphosarcoma are usually rather soft and tend to be of uniform size and distribution. Fever is rare except in the forms of the disease associated with acute lymphatic leukemia, in which it may be the most prominent symptom. The blood, however, frequently shows an increased percentage of lymphocytes, even in the absence of frank leukemia. Leukemia often accompanies lymphosarcoma or follows its development in children but is less frequent in adults.

Tuberculosis is extremely difficult to distinguish from Hodgkin's disease of any type, and it must be remembered that the two diseases occasionally occur in the same patient and, indeed, rarely in the same node. Redness of the overlying skin, fluctuation, caseation and sinus formation are not infrequent in tuberculosis, whereas they are extremely rare in Hodgkin's disease. It has been said that tuberculosis is prone to involve nodes in the anterior triangle of the neck, whereas Hodgkin's granuloma usually affects those of the posterior triangle, but this rule is treacherous. In our experience, large single nodes in the cervical or axillary region are likely to be tuberculous. The tuberculin reaction is usually positive in the presence of tuberculous lymph nodes, whereas it is almost always negative — even in high concentrations — in untreated Hodgkin's granuloma.

The presence of parenchymal disease of the lung should not be taken as proof that the entire process is tuberculous, for the pulmonary lesions of Hodgkin's granuloma may closely simulate those of tuberculosis, and the two diseases may coexist in the same person. Similarly, one should not be led astray by a past history of tuberculosis. Not infrequently proved tuberculous lymphadenopathy in childhood is followed by Hodgkin's granuloma in early adolescence or later life. The differentiation of these diseases can be easily made by biopsy.

Carcinomatous nodes are generally stony hard and are often fixed to the adjacent tissues, whereas the nodes in Hodgkin's granuloma, unless they have been heavily irradiated, are at most extremely firm. Carcinoma is likelier to cause pain, and the primary focus may be discovered and be patently cancerous. It must be remembered, however, that lymph nodes involved by rapidly growing carcinoma or sarcoma may be even softer than those of Hodgkin's granuloma, and that under these circumstances a clinical differential diagnosis may be impossible.

Chronically inflamed nodes secondary to a focus of infection that is often not obvious are not infrequently seen in children but are rare in adults. Small, pea-sized, painless, freely movable superficially situated nodes are, of course, often found in the cervical and inguinal regions of many healthy adults. Their presence need cause no necessary alarm, but the persistence of a notably enlarged node or group of nodes, especially if they are painless and fluctuate in size, calls for careful consideration whether a biopsy should be done. If there is any doubt, a node should be removed and the uncertainty thus removed. If there is only chronic inflammation, little has been lost; if the histologic picture is that of Hodgkin's granuloma, time has been gained. It must be constantly borne in mind that in many cases nodes affected by Hodgkin's granuloma fluctuate remarkably under the influence of coincidental nearby infection, and that the subsidence or even the virtual disappearance of cervical nodes should not be taken as proof that they merely represent chronic inflammation. This point is well illustrated by the following case.

R.S. (P13671), a 19-year-old girl, whose father had died of tuberculosis when she was a small child, in 1933 noted painless lymph nodes in the left side of the neck. For the next 5 years, these fluctuated greatly in size and varied in number, diminishing during the summer months and increasing during the winter, particularly with the occurrence of upper respiratory infection.

In January, 1938, the patient developed a severe "cold" and the nodes increased greatly in size. There was some cough and considerable dyspnea, but there was no disturbance of the general health or loss of weight. A node was excised and showed the typical picture of Hodgkin's granuloma. X-ray studies revealed the presence of a small mediastinal tumor.

Whether the nodes observed in 1933 were actually the site of Hodgkin's disease must, of course, remain an open question. Earlier biopsy would have decided this point, and earlier treatment — had the nodes been granulomatous — might have aborted the disease.

Infectious mononucleosis occasionally simulates Hodgkin's disease. Usually, however, the diagnosis is clarified by the peripheral blood picture and is substantiated by the heterophil agglutination test. Rarely the lymphadenopathy and splenomegaly of infectious mononucleosis remain for months after the subsidence of the acute disease. Under these circumstances, a careful history is important, and biopsy may be necessary.

Syphilis should cause no confusion if due attention is paid to the history and to the appropriate laboratory tests. We have seen a case, however, in which following a frozen-section diagnosis of lymphosarcoma a breast amputation and axillary dissection were done, the patient proving to have secondary syphilis. Parenthetically, it may be said that in malignant lymphoma a frozen section is an extremely slender reed on which to lean.

Lymphatic leukemia, rare in the twenties and thirties, is accompanied by the characteristic peripheral blood picture.

Rarely myelogenous leukemia is accompanied by generalized lymphadenopathy or enlarged nodes in a single region. The characteristic blood picture should make the differential diagnosis clear.

Very rarely tuberculosis causes generalized lymphadenopathy. The differentiation of the two cannot be made clinically. The possible confusion of Hodgkin's granuloma with erythema nodosum has already been referred to.

In most cases with systemic symptoms, Hodgkin's granuloma is accompanied even in its early stages by enlargement of superficial lymph nodes, but this may not be so, and in this event the diagnosis can often be made only by exclusion.

Fever may be the presenting and indeed the only symptom for weeks or months, and this sign will later be discussed in detail. Its presence, particularly if it is relapsing in type and is not obviously due to one of the more usual causes, should arouse the suspicion of Hodgkin's disease. A careful search for enlarged lymph nodes, especially in the abdomen, should be made, and x-ray studies may reveal the presence of involvement of the mediastinum. One of our patients had a relapsing type of fever for over a year prior to the development of any notable peripheral lymphadenopathy. True relapsing fever due to *Spirillum obermeieri* is practically never seen in this country. Undulant fever may now be diagnosed by appropriate laboratory tests. We have seen a case in which the presence of fever, cardiac murmurs, progressive anemia and a gradually enlarging spleen led to an incorrect initial diagnosis of bacterial endocarditis.

Rarely Hodgkin's granuloma begins with the symptoms of an acute upper respiratory infection — namely, chills, fever and cough. Such cases are almost always rapidly fatal. The tracheobronchial and mediastinal lymph nodes, the lungs and the pleura are frequently involved at the apparent onset. Usually, however, there is in addition the telltale superficial lymphadenopathy, either in the neck or in the axilla.

The differential diagnosis of mediastinal masses is always difficult. In our experience, a mediastinal tumor due to Hodgkin's disease is generally associated with enlarged cervical or axillary lymph nodes. The absence of such lymphadenopathy for any great length of time — two to six months — after the demonstration of the tumor is strong evidence that it is not due to Hodgkin's disease.

An adenomatous goiter, particularly if substernal, may cause diagnostic difficulties, for many of the symptoms and signs of hyperthyroidism, such as an increased metabolic rate, tachycardia, sweating, weakness, nervousness and even exophthalmos, may be present both in Hodgkin's disease and in other forms of lymphoma. If the question cannot be decided on clinical grounds, a determination of the organic iodine in the blood[1] will differentiate the two conditions, for in hyperthyroidism the iodine value is two to three times normal, whereas in Hodgkin's granuloma, even with an elevated metabolic rate, it is within normal limits.

Generalized itching, "eczema," weakness without other symptoms, abdominal pain, dyspnea, cough, hematemesis, melena, anorexia, amenorrhea, edema and a host of other nonspecific signs and symptoms testify to the protean character of the disease. In the presence of such symptoms, one can do no more than suspect that Hodgkin's disease is present and search for confirmatory evidence. Since well over 90 per cent of all cases eventually show enlargement of the superficial nodes, an opportunity to prove the diagnosis is usually at hand sooner or later.

Peripheral Blood Picture

Hodgkin's Paragranuloma

No significant change is seen in the peripheral blood picture in patients with Hodgkin's paragranuloma.

Hodgkin's Granuloma

Much has been written concerning the changes in the peripheral blood picture of Hodgkin's granuloma. Without question marked deviations from normal are frequent, especially in the late stages of the disease, but when one considers its protean manifestations and the number of organs that are involved, it is not surprising that there is no uniformity of opinion about just what these changes are. Out of the confusing medley of hematologic studies, however, certain facts stand out as being of practical importance. There is no truly characteristic change: that is, the disease cannot be diagnosed from a study of the peripheral blood alone.

A variable degree of hypochromic, or more rarely normocytic, normochromic anemia develops in a large proportion of cases, particularly in the late stages of the disease. In approximately one third of our cases — in all of which complete blood studies were done at each visit — the red-cell count eventually fell to 3,000,000 or lower. In 6 patients, 4 of whom were children, it was below 1,000,000. Children seem to be more liable to severe anemia than are adults. In 40 per cent of our patients under the age of twelve years, the red-cell count fell below 2,000,000, in contrast to 13 per cent of the entire group. The pathogenesis of this anemia is uncertain. In some cases it is apparently dependent on involvement of the bone marrow but that this is not invariably so is attested by the facts that in the patient in our series who had by far the most extensive bone lesions the count never fell below 3,600,000 and that in another patient who had a terminal count of 500,000 there was no evidence of bone involvement. In rare cases,[2, 3] there is hemolytic anemia of considerable severity, simulating in some respects, in so far as the peripheral blood is concerned, that of a congenital or acquired hemolytic anemia. In many of these cases there is a pseudomacrocytosis due to the presence of an increased number of reticulocytes. There may or

may not be alteration in the fragility of the red cells. The exact explanation of this hemolytic anemia in Hodgkin's granuloma is yet to be determined.

It is generally agreed that although the white-cell count is rarely normal throughout the disease, leukocytosis is extremely frequent, and that, on the other hand, extreme leukopenia may occur. Sixty-three per cent of our patients had a white-cell count of 11,000 or over. In 12, the count was at one time or another above 25,000. The leukocytosis occurs quite independently of fever. Marked leukopenia occurred in 12 per cent of the cases. There seemed to be no correlation between the clinical findings and the leukopenia, but in certain cases the latter seemed to be due to irradiation, especially when this had been applied to the abdomen. Marked fluctuations in the white-cell count may occur within a short period of time and without apparent cause.

Certain significant changes take place in the differential white-cell count. By far the most constant one is a relative or absolute increase in the percentage of polymorphonuclear neutrophils, a point on which all investigators are agreed. In 81 per cent of our cases, this percentage was at one time or another over 75; in 5 cases, it was 95 or more. This change tends to become increasingly more pronounced as the disease advances. There is a strong tendency toward a shift to the left, and myelocytes in varying proportions are not rare. They were found, usually in small numbers and inconstantly, in 11 per cent of our cases.

An occasional case shows an increased number of eosinophils. Baldridge and Awe[4] report a case with a white-cell count of 43,000 and 83 per cent eosinophils. Uddströmer[5] found over 6 per cent eosinophils in only 12 per cent of his cases, and was unimpressed either by the importance of eosinophilia in the disease or its relation to itching, which has been stressed by many writers. In our experience, marked eosinophilia is rare, and when one recalls the large variety of conditions accompanied by an increase of these cells, it seems not improbable that in certain cases, at least, the eosinophilia is unrelated to the Hodgkin's granuloma.

Monocytes are frequently increased in number, and in rare cases greatly so.

The number of platelets shows no constant change, although they are not infrequently moderately increased.

In summary, the most frequent blood changes in Hodgkin's granuloma are a normochromic or hypochromic anemia, a moderate increase in the white-cell count and a moderate to great increase in the percentage of polymorphonuclear neutrophils.

Hodgkin's Sarcoma

A moderate degree of anemia — usually hypochromic, occasionally normocytic and rarely macrocytic — is seen in Hodgkin's sarcoma. In only a small percentage of the cases, however, does the anemia become extremely marked. In 5 of our cases, the red-cell count was below 2,000,000, with a correspondingly low hemoglobin. One of these patients may possibly have had a hemolytic type of anemia, for the icteric index was 50 and there was nothing to account for the jaundice. Fragility tests were not done, however, so that one cannot be categorical on this point.

The white-cell count is usually normal; rarely it is moderately elevated. The percentage of polymorphonuclear neutrophils is almost invariably high, and in approximately one third of the cases there is a marked increase in the percentage of monocytes. Myelocytes occasionally occur.

There seems to be no correlation between the hematologic findings and either the organs involved or the course of the disease.

Fever

Hodgkin's Paragranuloma

In patients with Hodgkin's paragranuloma, fever does not occur.

Hodgkin's Granuloma

Fever of one type or another, unaccompanied by sepsis or incidental and unrelated infectious diseases, is an extremely frequent finding in Hodgkin's granuloma. Uddströmer[5] in Sweden reported that fever was present in 54 per cent of 484 collected cases. In our series, it occurred at one time or another in at least 43 per cent; it is probable that the incidence would have been higher if every patient had been constantly observed throughout the course of the disease.

The fever may be of various sorts. Most frequently it is, as Uddströmer puts it, "typeless." Occasionally it is intermittent or remittent, reaching 101 to 105°F. every day. More rarely a high continuous fever is seen. Such a finding is almost always terminal. It is common knowledge that pyrexia becomes increasingly frequent as the disease advances.

During febrile periods unassociated with sepsis, it is usual for the lymph nodes to increase in size; splenomegaly, if it exists, frequently becomes more marked, and the patient is generally prostrated, weak and debilitated. Tachycardia, sometimes extreme, almost invariably accompanies the fever. Occasionally, however, one sees a patient who, in spite of a moderate increase in temperature — 99 to 101°F. — feels perfectly well and is, indeed, unaware of any febrile reaction, and the fact that peripheral lymphadenopathy is absent or inconspicuous at the time may render the correct diagnosis extremely difficult.

Infections, particularly those affecting the upper respiratory tract, are prone to cause coincident enlargement of lymph nodes close to the affected

area. Too often such lymphadenopathy is regarded as due to the infection alone; it is too infrequently recognized that lymph nodes involved by Hodgkin's granuloma but not greatly enlarged may rapidly increase in size after what appears to be a simple infection.

There is a peculiar and characteristic reaction that deserves special attention — so-called "Pel-Ebstein fever." In 1887, Pel[6] and Ebstein[7] independently described a relapsing form of fever characterized by periods of pyrexia, alternating with intervals during which the temperature is normal or subnormal. The febrile periods are marked by a gradual daily increase in temperature to 104°F. or more and a subsequent equally orderly decline to normal. The pulse and respirations usually follow the temperature in such a manner that when the latter is 104°F. the pulse is 120 to 150 and the respirations are 30 to 35. During the period of increased temperature, most patients feel extremely ill. There follows an afebrile period of weeks or months, during which the patient may return to apparently perfect health, but sooner or later the fever returns and the afebrile intervals become shorter, and eventually the fever becomes continuous.

This syndrome, although usually referred to as Pel–Ebstein fever, was accurately described by Murchison[8] in 1870 in a case of so-called "lymphadenoma"—the British term for Hodgkin's granuloma. By rights, if any man's name is to be attached to the syndrome it should be Murchison's.

Mac Nalty[9] has made a special study of the fever in Hodgkin's disease.

The following case is illustrative of Pel–Ebstein (Murchison) fever.

F.C. (P2903), a 22-year-old house painter, entered the hospital on January 20, 1931. In the spring of 1930, he noted a painless lump the size of a walnut in the left side of the neck. This was removed at another hospital and showed the usual histologic picture of Hodgkin's granuloma. At that time there were no other symptoms, and the patient was lost sight of. In October, 1930, he began to have attacks of fever, malaise and anorexia. These lasted for 10 days or so and recurred after an interval of apparent good health, about every 6 weeks. During each attack the patient was confined to bed and extremely prostrated, but between attacks he felt perfectly well. Each febrile episode was ushered in by a marked sense of drowsiness and anorexia, and the temperature rose higher each night until by the end of 5 or 6 days it had reached 104 or 105°F. It then gradually subsided to normal by about the 15th day. During the febrile period there was marked anorexia, sweating and extreme prostration. As soon as the temperature became normal the patient again felt quite well and was able to resume his daily work.

Physical examination on entry showed no abnormalities except for a small scar in the left side of the neck. An x-ray film of the chest showed a slight increase in the mediastinal shadow. There was slight anemia. The white-cell count was normal but the differential count showed 90 per cent polymorphonuclear leukocytes and 10 per cent lymphocytes.

Following x-ray therapy to the chest the small mediastinal mass disappeared, but there was no symptomatic improvement, and the patient continued to have recurrent attacks of fever, malaise, extreme weakness, anorexia and fleeting generalized pains. The intervals between attacks constantly became shorter, and the fever finally became continuous. X-ray therapy had no effect, and Fowler's solution in large doses caused no improvement.

Four months after entry, the liver and spleen became palpable. One month later, jaundice appeared and the spleen reached the umbilicus, but there was still no peripheral lymphadenopathy. Anorexia was extreme and loss of weight very marked, and drowsiness became a more and more prominent feature. The patient's strength rapidly failed, and he died on April 17, 1931, about 7 months after the onset of the generalized symptoms and 1 year after the first symptom of painless lymphadenopathy

Autopsy. Post-mortem examination showed widespread Hodgkin's granuloma, with scarcely an organ spared. The main lesions were chiefly in the liver, the spleen and the retroperitoneal and mediastinal lymph nodes.

Uddströmer[5] found this type of fever in 7 per cent of his cases; in our series it was present in 5 per cent.

Hodgkin's Sarcoma

Fever occurs in approximately one third of the cases of Hodgkin's sarcoma, although it follows no particular pattern and is rarely very high; not infrequently, however, one sees a septic type of temperature reaching a daily peak of 102°F. Pel–Ebstein (Murchison) fever does not occur. Definite chills are occasionally seen, and in patients having them the prognosis is extremely bad. We have never had such a patient who lived more than two years after the apparent onset of the disease.

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MENTAL BREAKING POINTS*

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IF THE psychiatrist interviews servicemen who have broken down mentally day after day, month in and month out, he is likely to conclude that all men have mental breaking points; furthermore, he may wonder how it is possible that the United States is winning the war. His outlook on life and his interpretation of human behavior are apt to be colored, if not strongly influenced, by the symptoms and diseases that he finds in his patients. It is evident that all patients referred to the psychiatrist in a military hospital must have mental symptoms of some type. To decide that all men have mental breaking points on the ground that all those whom the psychiatrist interviews have shown one, or have had nervous breakdowns, appears to be drawing general conclusions from an extremely small and badly chosen sample.

In the past year the popular press has issued many warnings and even appeals to the public, and in particular to the families and friends of servicemen, on what to do or say, or what not to do or say, when veterans come home. Most of this advice is sincere but misdirected. If applied to those who have developed nervous or mental disorders it might be of more value, although one should avoid generalizations and should treat each man according to his needs. The reaction against this type of advice among men in the service who are in good physical and mental health is in many cases one of resentment and annoyance. It is believed that the basic reason why the majority of returning veterans are disturbed by the special treatment accorded them by their family and friends is that they are getting the treatment intended, not for them, but for the psychiatric war casualties, among whom they do not belong.

Why should this error occur? The explanation is thought to lie in the belief or theory, held by laymen and by physicians alike, that all men have mental breaking points or can take only so much, or, to put it another way, that all men are apt to "crack up" or have a nervous breakdown if the stress of combat or military life is severe enough.

One not infrequently hears such a remark as "It was more than he could bear," "A man can only take so much," or "He couldn't take it any longer." These statements are widely used by laymen to denote a nervous breakdown in the face of overwhelming misfortune or severe environmental stress. The emphasis is frequently placed on "an unkind fate" or on "outrageous fortune," over which the individual has no control. The victim is supposedly caught in the toils of circumstance and irresistibly crushed. Since World War I considerably more attention has been paid to the individual than to the environmental stress, and there appears to be an unofficial but fairly general agreement among psychiatrists in the armed forces that the stability of a man's personality prior to entry into the service is more important in the maintenance of mental equilibrium than is the severity of the combat stresses to which he may have been subjected.

Obviously, the terms "breakdown" and "breaking point" have physical and mechanistic origins and are used only metaphorically — but nevertheless effectively — in referring to mental disorders. Such terms as "stress," "tension," "pressure," "weight," "force" and "impact" have been borrowed because they are descriptive, and because analogies can be easily drawn, supported or reinforced by their use. Psychiatrists use the term "nervous breakdown" with caution, preferring some such descriptive or diagnostic term as "psychoneurosis," "dementia praecox" or "psychopathic state" — to mention only a few of these — denoting the character or trend of the disorder. These terms are labels, at times hardly more satisfactory — and certainly more confusing to the layman — than "nervous breakdown." It is evident that any expression of this kind must be related to some frame of reference that indicates inefficiency or failure in performance, such as unfitness for military duty, for life in the community — involving mental institutionalization — or for work. In each case the essential element is a failure of performance or loss of efficiency for the task assigned, which may vary both qualitatively and quantitatively. For practical purposes in this paper, the term "nervous breakdown" refers to such failure of efficiency that is directly or indirectly related to an emotional or mental instability that necessitates medical care. So long as such a man remains a patient in a medical unit, he is an ineffective person so far as the military service is concerned.

Such a definition of nervous breakdown obviously includes a wide range of mental disorders, from low morale, with its aversion to fighting and hardships, to major psychoses. It is also applicable to those self-centered persons who are feigning or exaggerating various somatic complaints to escape military duty. It is evident to many neuropsychiatrists working in military hospitals that a great many patients on medical, surgical and psychiatric wards suffer from a certain lack of morale or failure in performance that is not dependent on unconscious mental conflicts and should certainly not be

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classified as a psychoneurosis. To say that these patients are not psychiatric cases because there is no name for them except "gold brickers," "inadequate personalities" or "opportunists" is to neglect a serious problem, for the number of hospital beds occupied by this type of patient is large, and will be proportionately larger in veterans' hospitals after the war.

The definition under discussion need not exclude patients who suffer from some organic disorder but whose incapacitating symptoms are primarily nervous or emotional. It is evident that many situational and symptomatic disorders of this kind masquerade under such terms as "gastritis," "colitis," "effort syndrome," "myositis," "rheumatism," "eye strain," "neuritis" and "neuralgia" — to mention only a few. The inefficiency and failure of many of these persons are frequently more closely related to emotional instability or lack of tolerance of the military situation than to defective functioning of the organ in question. This broad definition of nervous breakdown may be objected to as being all-inclusive, and it is admitted that there are many men on the wards in military hospitals who would probably never consult a physician in peacetime, unless it were to obtain an excuse for inability to carry out life's obligations in a satisfactory manner. Such patients' disability or failure in performance is largely one of loss or lack of morale. They constitute casualties fully as much as do men with wounds, and in addition they have a much more damaging influence on the morale of the group to which they belong.

The retreat of the British from Mons in World War I is cited as an instance of stress so severe that many broke under the strain.[1] One should, however, not lose sight of the fact that appreciable numbers did not break down — notably those who stood, fought and won in the Second Battle of the Marne. In this war it would be of considerable interest and value to know the actual percentage of nervous breakdowns among those who were evacuated at Dunkirk, among the men who fought on Guadalcanal or among the troops engaged at Stalingrad, Anzio, Dieppe, Tarawa and Iwo Jima. If one happens to see a large number of neuropsychiatric patients from the same combat zone, one is apt to conclude that these casualties were unusually high, whereas actually one may know little of the total number of troops involved.

One cannot base a diagnosis of mental disorder entirely on the symptoms presented. The patient's attitude toward them is far more important than their degree of severity. One sees many men returning from combat or operational duty and many survivors of ship sinkings who are anxious to carry on in the face of distressing somatic symptoms. This has been observed both in officers and among the enlisted personnel. The essential feature seems to be a high morale or sense of duty, dependent primarily on the stability of the personality prior to entry into the service. The presence or absence of morale in an individual or a group is a complicated matter. Morale depends on many factors, two of which appear to be of outstanding importance. One is the inherent emotional stability of the person; the other is his quality of leadership or confidence in leadership. There are many men with a defective emotional adjustment who are held in line by the *esprit de corps* of the group, being profoundly influenced by leadership, who break down when this protective influence is removed or regain their morale if effective leadership is provided. It must be borne in mind, however, that a large percentage of men in a given unit do not break down mentally, irrespective of what may happen to their leaders. It is true that the military effectiveness of any unit may be gravely influenced by a relatively small percentage of casualties of whatever type, and particularly by mental casualties. If competent, skilled and resourceful leaders take over such a demoralized unit, their task is not so hopeless as might appear at first glance, since they are dealing with a group that has had its most unstable men screened out by exposure to combat, so that those who remain, together with replacements, have not had nervous breakdowns. This is not meant to detract in any way from the role of leadership in producing an effective fighting unit or to oversimplify the problem of the maintenance of morale. It is intended only to point out that at times too much attention is focused on the man who breaks down mentally. An entire unit may be branded as "NP" or "psycho" even though the unit has a relatively low mental-casualty rate, the men who have stood up under all kinds of trying circumstances being lost sight of. The emotionally unstable are evacuated to safer zones and are eventually returned home, the majority being discharged from the service with varying degrees of disability. For the larger percentage of men who did not break down, "Valor-like virtue is its own reward" and may turn out to be a reward full of bitterness in the postwar period, if society fails to provide adequate opportunity for the veterans who are chiefly responsible for the winning of the war.

It has been the practice at this center, when examining sailors from various fighting ships or marines who had been through a strenuous campaign, to ask whether they knew of anyone aboard their ship or in their outfit who broke down mentally. The questions were asked both of men who showed evidence of emotional disorder and of those who did not. The replies appear to be of some significance. Without exception these men knew of only one or two others who had suffered a breakdown, and in some instances an entire ship's crew or company of marines had no psychiatric casualties.

It is realized that this method of acquiring data will not bear critical evaluation, since a fair number

of men have a delayed reaction and may not develop their mental disorder until later, and since the casual observer is apt to mistake the true diagnosis in certain cases or to misinterpret unusual behavior. It is, however, not necessary to rely on such hearsay, particularly when more reliable information is available. The psychiatrist at a naval receiving station has an unusual opportunity of interviewing all the survivors of sunken ships and all men returning from combat or naval operations. The type of screening thus accomplished is unlike any other, but most closely resembles that done at induction centers, with an important difference: all men interviewed had been subjected to the test of combat or overseas duty. It can hardly be said that they had not been subjected to the type of stress that precipitates a nervous breakdown in susceptible persons.

The significant fact concerning these men returning from overseas is that so few of them showed any evidence of emotional instability or nervous breakdown. The great majority declared that they had never experienced any disabling nervous symptoms, even though many had been in combat on a number of occasions. It was true that the majority admitted that they had experienced fear and its attendant autonomic symptoms, but exceptionally few had been incapacitated by fear.

Two thousand men were given a questionnaire, the Cornell Selectee Index,[2] a short psychiatric or psychosomatic inventory consisting of sixty-four questions designed to elicit evidence of emotional instability. In addition, each man was interviewed. Of these 2000 men, 500 were survivors of sunken ships, 1000 had returned from combat duty, and 500 were being returned from overseas duty, many of the latter from advanced outposts, where the stress of military duty is said to be even more trying than that of actual combat. Only 10 showed evidence of nervous breakdown according to the definition given earlier in this paper. These men showed evidence of emotional instability, with an increased startle reaction, increased fatigability, tremors, palpitation, insomnia, nightmares, headache, irritability, gastrointestinal disturbances, anxiety and depressive reactions, severe enough to render the victim unfit for military duty and to warrant sending them to a naval hospital.

Table 1 shows the percentage of each group of men giving twenty-five or more positive answers. It is claimed that this method of scoring screens those having a severe degree of emotional maladjustment. It is of interest that although 5 per cent of the 2000 men showed evidence of severe nervous symptoms, all but 10 of these had been able to serve in the armed forces overseas without developing evidence of a nervous breakdown. It would be erroneous to assume that these 90 men had been missed in answering this index. Personal interviews ruled out this factor and established a previously mentioned principle, namely, that symptoms alone are not indicative of a nervous breakdown, the subject's attitude and reaction toward them being far more important than their severity

This low percentage of men with a significant nervous disorder was at first somewhat startling, since the psychiatrist had rarely had an opportunity to interview men who had been through combat or the prolonged stress of trying overseas conditions and had continued to maintain their mental equilibrium. It may be argued that a certain number of men had been hospitalized for nervous disorders prior to the return of their units to this country, and also that some of those interviewed may be expected to develop disabling nervous symptoms many weeks or months after their experience; but even allowing for these cases, the percentage of men who develop a nervous breakdown under the stress of military service is extremely small if compared to the total number in the service. This group of psychiatric war casualties should be adequately cared for, and those familiar with the military services know that such rehabilitation programs are under way at the present time.

TABLE 1. *Cornell Selectee Index Scores of Returned Personnel.*

GROUP	NO. OF MEN	PERCENTAGE WITH SCORES OF 25 OR MORE
Men returned from combat duty....	1000	6
Men returned from noncombat duty.	500	6
Survivors of sunken ships.....	500	4
Total.....	2000	
Hospitalized for nervous breakdown .	10	100

It is believed that there has been considerable overemphasis on the serious consequences to the man who has been in combat or under the prolonged stress of military operations. It is not the intention of this paper to minimize the important work done by psychiatrists in the detection, elimination and care of psychiatric war casualties. It is thought, however, that a complete over-all picture of the mental health of the men in the armed forces is not reflected by the reports in the popular press nor by those emanating from military hospitals, for it is evident to psychiatrists serving in such military activities that they see only those who have broken down mentally or have failed in the performance of their duty, and thus tend to lose their perspective regarding the mental health of all men under arms. It should be apparent that even though the definition of nervous breakdown given above is broad, the percentage of men in the service who have had such a disorder is relatively small.* This is due in part to the excellent work done by psychiatrists at induction centers and training stations — frequently working under considerable handicap — in the elimination of the psychiatrically unfit. The great majority of those

*Estimated 5 per cent at the maximum.

retained have repeatedly proved themselves to have stable personalities.

* * *

It is obvious that not all men suffer mental breakdowns under severe stress. It is not meant to imply that the human organism is indestructible, since physical exhaustion and organic disease are bound to take their toll, or that a person under prolonged and severe stress may not fail in some manner other than mental. To argue whether a stable personality develops a nervous breakdown if just the proper set of stresses happen to strike at the same time is profitless. But this is not a question of practical importance, since experience shows that the great majority of fighting men are capable of absorbing the shock of all the blows to which they are subjected without showing signs of a nervous breakdown.

It would be erroneous to judge the mental health of all men under arms by the picture presented on the neuropsychiatric wards of military hospitals. It would be just as erroneous to assume that the 10 men out of 2000 cited above represented all those who may subsequently break down mentally under service conditions. It is apparent that men with stable personalities are much less susceptible to the stress of military service in wartime than are those having poorly balanced or defective personalities. It is principally the man with a defective or maladjusted emotional life who is likely to break mentally under the stress of military duty, and although it is generally believed that even the most stable personality may crack under the strain of combat, this state of affairs is exceedingly rare. All that can be said with certainty is that only those who actually break have mental breaking points, and that experience at a naval receiving station in examining men fresh from combat and noncombat duty and survivors of ship sinkings does not support the widely accepted theory that all men have mental breaking points.

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THE INCIDENCE OF LEUKEMIA IN RADIOLOGISTS*

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EXPOSURE to x-rays has for some time been regarded as a possible cause of leukemia. This belief is based in part on results obtained by experimental exposure of animals to x-rays[1–4] and in part on several reports of cases of leukemia occurring in workers exposed to radiation.[5–15] Definite proof of such an etiologic relation has not been established, however, and some authors have expressed doubt concerning it. Thus, Evans and Roberts[8] stated in 1928, after a review of the literature to that date, "Although the possibility of causal association cannot positively be denied, the evidence is not conclusive." Haagensen[9] wrote, "Doubt may be raised as to the relationship of the exposure to radiation and the development of leukemia," and Warren and Dunlap,[16] in their comprehensive review published in 1942, stated: "Few examples of leukemia have been described in persons chronically exposed to radiation. Only 24 case reports are found in the literature if one excludes all reports of leukemia following the therapeutic irradiation of lymphatic tumors."

More recently, Henshaw and Hawkins,[17] having found that none of the existing evidence furnishes any direct proof that radiation actually acts as a carcinogenic agent in the induction of leukemia in human beings, determined the incidence of leukemia in physicians from the death notices published weekly in the *Journal of the American Medical Association* and compared it with similar data regarding the general population, derived from the vital statistics of the United States Bureau of the Census. They found that during a ten-year period (1933–1942) 0.53 per cent of physicians died of leukemia, as compared with 0.39 per cent of the general population. After making corrections for differences of age, sex and other factors that influence the outcome, they arrived at the following conclusion: "Leukemia was recognized approximately 1.7 times more frequently among physicians than among white males in the general population." They stated that, although these observations furnished no direct proof that radiation acts to incite leukemia in human beings, they were nevertheless in accord with the findings on experimental animals in which exposure to x-rays had been found to increase the incidence of leukemia.

Since the majority of physicians are not subject to exposure to radiation, it seems that a comparison of the incidence of leukemia among radiologists and that among other physicians should give more conclusive results. The present report is based on a statistical study of deaths of physicians reported in the *Journal of the American Medical Association* during the ten-year period 1935–1944.

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It must be admitted that the results cannot be perfect, first, because, as indicated by Henshaw and Hawkins,[17] the cause of death is not given in about 25 per cent of the obituary notices, and second, because in an even greater number there is no indication of possible x-ray exposure. For example, some of the physicians not listed as radiologists but dying from leukemia may have been exposed to radiation, — the case reported at the end of this article is of this type, — which would increase the incidence of leukemia in the exposed group. Conversely, some unexposed physicians, the cause of whose death is not given, may have died of leukemia, which would increase the incidence in this group. It is not unreasonable, therefore, to assume a tendency of the various unknown opposing factors to balance each other. At any rate, if the difference between the two groups is sufficiently large, it should not be significantly affected by these discrepancies.

From 1935 to 1944, inclusive, the deaths of 34,626 physicians were recorded in the *Journal of the American Medical Association*. Two hundred and five of these men were listed as radiologists. The total stated number of deaths from leukemia is 158. Eight of these occurred among the 205 radiologists, giving a percentage of 3.9; 150 were among the 34,421 physicians not listed as radiologists, a percentage of 0.44. Therefore, those known to have been exposed to radiation had an incidence of leukemia more than eight times as great as those who are not listed as radiologists. Although this difference may be affected somewhat in either direction by the previously discussed lack of complete information, it seems too great to be wiped out or to be significantly modified.

This statistical study, which supplies substantial evidence that exposure to radiation is a potential cause of leukemia, was suggested by the occurrence of this disease in a physician who had repeatedly been exposed to radiation over a long period of time. The following data summarize the findings in this case.

R.J., a 51-year-old dermatologist, had for several years failed to take precautions against exposure to radiation that he used for treating his patients. Symptomatically his illness began in the fall of 1941, the major complaint consisting of pain in various parts of the body, especially in the chest. It became quite severe during a period of 6 weeks and was aggravated by motion and deep respiration. Slight fever (99.2°F.) was present. The skin was pale and suggested anemia, which was confirmed by examination of the blood, the red-cell count being 2,610,000 and the hemoglobin 45 per cent. Leukopenia was present, the number of leukocytes varying between 1500 and 5540 per cubic millimeter and the percentage of neutrophils between 4 and 54. The lymphocytes, largely normal in appearance, were the most numerous leukocytes in the majority of smears. At times occasional immature cells of the lymphoblastic type were found. The leukopenic blood picture persisted essentially unchanged for 6 months, when the total number of leukocytes began to increase and reached a maximum of 163,200 within 2 weeks, with a differential count typical of lymphatic leukemia. The patient died on August 20, 1942, approximately 9 months after the onset of symptoms. Autopsy confirmed the diagnosis of lymphatic leukemia.

This case is intentionally omitted from the group of 8 cases of leukemia occurring in radiologists, because the notice of this patient's death gave no indication that he had been exposed to radiation. If his case were included in the radiologic group, the percentage of leukemic deaths therein would be increased to 4.3, and the percentage in the nonradiologic group, in which his case was included, would be slightly lower, thus accentuating the difference between them.

The fact that the patient was a dermatologist suggested a separate estimation of the incidence of leukemia in physicians practicing that specialty. During the ten years covered by the study, 60 physicians listed as dermatologists died. Two of them, including the one reported in this article, died of leukemia, giving a percentage of 3.3. This approaches the incidence in the radiologic group, but the number of cases is too small to be of statistical significance.

Summary

A statistical study of 34,626 obituary notices in the *Journal of the American Medical Association*, covering the ten-year period from 1935 to 1944, reveals that the incidence of leukemia among 205 physicians listed as radiologists was 3.9 per cent, which is more than eight times as great as the incidence (0.44 per cent) among those not listed as radiologists.

This marked difference is substantial evidence that exposure to radiation is a potential cause of leukemia.

A case of lymphatic leukemia in a dermatologist who failed to take adequate precautionary measures against radiologic exposure is reported.

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ICE CREAM AS A SOURCE OF RIBOFLAVIN, CAROTENE AND ASCORBIC ACID*

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MANY people consider ice cream as a luxury food, a dessert or an attractive adjunct to teas and other social occasions where light refreshments are served. Consequently, this food does not ordinarily receive proper recognition as a valuable source of protein, fat, carbohydrate, minerals and vitamins, all of which are essential dietary constituents. In fact, most people fail to realize the extent to which ice cream is consumed in this country. It is reported that 412,144,000 gallons were used during 1943,[1] or over 3 gallons for every man, woman and child. It is difficult to estimate accurately the retail value of this ice cream, since large quantities are retailed in cones of various sizes and as individual servings at stores and resorts, but on the basis that ice cream retails for 75 cents a quart, the retail value of ice cream consumed in 1943 amounted to at least $1,236,432,000. This is about one fourth the retail value of the whole milk and over twice the retail market value of the eggs, apples or potatoes consumed annually.[2] A survey of the literature, however, failed to reveal much information concerning the vitamin content of this valuable food, and this study was conducted to accumulate data in this connection.

PREPARATION OF ICE CREAM

The coffee, maple and vanilla ice creams used in this study were made at the Dairy Manufactures Laboratory, under the conditions maintained for training students and for the preparation of ice cream for the Massachusetts State College dining halls. The ingredients for 100 pounds of ice-cream mix were 24.4 pounds of cream (40 per cent), 56.3 pounds of whole milk, 15.0 pounds of cane sugar, 4.0 pounds of skimmed milk powder, and 0.3 pounds of gelatin. The whole milk and cream were produced by the college herd of Ayrshire, Guernsey, Holstein, Jersey and Shorthorn cows fed and maintained under conditions similar to those of local progressive commercial dairies.

The ingredients were thoroughly mixed and pasteurized by the holding method at 145°F. for thirty minutes. When pasteurization was completed, the mix was passed through a homogenizer at 145°F. under 2500 pounds pressure per square inch to break up the fat globules and obtain a product of uniform composition. The ice-cream mix was cooled to 40°F. and allowed to blend or ripen for eighteen hours. At this stage of manufacture, the ice cream weighed 9.25 pounds per gallon. The mix was then placed in a batch freezer, and flavoring was added at the rate of 6 ounces of vanilla, 32 ounces of coffee extract or 12 ounces of imitation maple flavor per 100 pounds. During the freezing process the flavoring was intimately incorporated and sufficient air was whipped into the mix so that the final ice cream weighed 5 pounds a gallon. Thus, essentially half the volume of the finished ice cream consisted of occluded air. The finished ice cream was placed in commercial 4-ounce cardboard containers and held in a refrigerator at −10°F. This ice cream was not strictly comparable to the present-day wartime commercial product, since the latter ordinarily contains only about 10 per cent of fat, whereas the ice cream used in this study contained 12 per cent of fat.

ASSAY PROCEDURES

Carotene

The carotene content of the ice cream was determined by a modification of the method of Boyer, Spitzer, Jensen and Phillips.[3] Briefly, the procedure was as follows. A 20-gm. sample of ice cream was extracted with 30 cc. of alcoholic potassium hydroxide solution — 40 gm. of potassium hydroxide completely dissolved in 50 cc. of distilled water and thoroughly shaken with 180 cc. of ethyl alcohol — and allowed to stand for three hours. Twenty-five cubic centimeters of diethyl ether was added and the mixture was vigorously shaken for one minute. After the second extraction with ether, the two extracts were combined, 25 cc. of Skellysolve B was added, and the mixture was vigorously shaken for fifteen seconds. Twenty-five cubic centimeters of 80 per cent methyl alcohol was added and the mixture was shaken for one minute. The alcohol-water-ethyl ether layer was discarded. Fifty cubic centimeters of 3 per cent sodium sulfate solution was added and the mixture was vigorously shaken four separate times. The water layer was discarded. Ten cubic centimeters of Skellysolve B was added and the Skellysolve layer was filtered through No. 41 Whatman filter paper containing about 5 gm. of anhydrous sodium sulfate into a 50-cc. amber flask. The separatory funnel was rinsed with Skellysolve, and the filter paper and sodium sulfate were washed with separatory rinsings until a volume of 50 cc. had been reached. The regular procedure was then followed.

Riboflavin

The following procedure was used to determine the riboflavin content of ice cream. Ten grams of ice

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cream was extracted for one hour with 50 cc. of acetone-sulfuric acid solution (3 parts acetone to 1 part of 1.0N sulfuric acid). After extraction acetone was added to 60 cc. (the original volume), and 6.5 per cent trisodium phosphate solution was added until the reaction of the mixture was pH4.5. Sufficient distilled water was added to make 200 cc. Fifty cubic centimeters of subnatant liquid was withdrawn, and to this there was added 7 to 10 drops of 4 per cent potassium permanganate solution and sufficient 1 per cent hydrogen peroxide to decolorize. The volume was made up to 100 cc., and the solution was filtered. Three 20-cc. aliquots, designated as A, B and C, were withdrawn. One cubic centimeter of water was added to aliquot A, 1 cubic centimeter of standard riboflavin solution (.0025 mg. per cubic centimeter) to aliquot B and 1 cc. of water and a few crystals of sodium hydrosulphite to aliquot C. Ten cubic centimeters of each aliquot was transferred to cuvettes and read in the spectrophotometer, as described by Holmes, Jones, Wertz and Kuzmeski.[4]

Reduced Ascorbic Acid

The ascorbic acid content of the ice cream was determined by a modification of the method of Holmes, Tripp, Woelffer and Satterfield.[5] Twenty-five grams of ice cream was weighed into a tared centrifuge tube, intimately mixed with 25 cc. of a mixture of equal parts of 5 per cent metaphosphoric acid and 10 per cent acetic acid, and centrifuged for ten minutes. The mixture was filtered and repeatedly washed with 5 per cent metaphosphoric acid. Approximately 50 cc. of the filtrate was transferred to a 125-cc. Erlenmeyer flask, 0.5 gm. of 300-mesh Bentonite was added, and the mixture was shaken vigorously for one minute, allowed to settle and filtered. Ten-cubic centimeter portions of the clear, colorless filtrate were titrated with a standard 2-6 dichlorophenolindophenol dye solution.

Results

Ten samples of coffee, of maple and of vanilla ice cream were assayed for carotene, riboflavin and reduced ascorbic acid. The results were quite uniform for all three flavors. The extreme values for carotene and riboflavin for each of the three flavors are reported below, but since close agreement was obtained for the different samples of the flavors, only the average values are reported in Table 1.

The carotene content of the coffee ice cream varied from 0.09 to 0.10 mg. per 100 gm., with an average of 0.09 mg. The amount of carotene in the maple ice cream ranged from 0.11 to 0.12 mg. per 100 gm., with an average of 0.12 mg. The vanilla ice cream contained from 0.09 to 0.10 mg. per 100 gm., with an average of .09 mg. Thus, the average values for the carotene content of coffee and of vanilla ice cream were identical, but that of maple ice cream was somewhat higher.

The riboflavin content of the ice cream was virtually uniform for each flavor. The amount in the coffee ice cream varied from 0.25 to 0.29 mg. per 100 gm., with an average of 0.27 mg. The maple ice cream contained from 0.24 to 0.28 mg. per 100 gm., with an average of 0.26 mg. The amount of riboflavin in the vanilla ice cream ranged from 0.25 to 0.29 mg. per 100 gm., with an average of 0.26 mg.

Table 1. *Vitamin Content of Ice Cream.**

Experiment No.	Carotene	Riboflavin	Reduced Ascorbic Acid
	mg./100 gm.	mg./100 gm.	mg./100 gm.
1	0.10	0.27	0.00
2	0.10	0.26	0.00
3	0.11	0.26	0.00
4	0.10	0.27	0.00
5	0.10	0.28	0.00
6	0.10	0.26	0.00
7	0.11	0.26	0.00
8	0.10	0.26	0.00
9	0.10	0.25	0.00
10	0.10	0.27	0.00
Averages	0.10	0.26	0.00

*Averages for ten samples of coffee, maple and vanilla ice cream.

The reduced ascorbic acid was determined for ten samples of each of the three flavors. Only five or six of the thirty samples showed even a detectable trace of reduced ascorbic acid. These results were not unexpected, however, since, as stated above, nearly half the volume of the finished ice cream consisted of air that had been whipped into it during the freezing process. The air dispersed throughout the ice cream obviously favored rapid oxidation of the reduced ascorbic acid.

Obviously, the amount of carotene, riboflavin and reduced ascorbic acid in any ice cream is influenced by the type and amount of the materials used in its manufacture, the procedure used for preparing it and the conditions under which it is stored during the interval between manufacture and consumption. Since these factors vary widely with conditions and localities, the nutritive value of both homemade and commercial ice cream is quite variable.

Discussion

It is interesting to compare the amount of carotene found in the three flavors of ice cream — 0.09 mg., 0.12 mg. and 0.09 mg. per 100 gm. — with that found in some other common foods. The amount in fresh whole milk is apparently quite variable, for Tuzson[6] found 0.003 mg. per 100 gm., and Guggenheim[7] reported 0.028 mg., 0.017 mg. and 0.010 mg. per 100 gm., respectively, for milk produced in hill, valley and urban areas of Palestine. Olson, Hegsted and Peterson[8] state that fresh whole milk contains 0.021 mg. of carotene per 100 gm., and Dornbush, Peterson and Olson[9] found 0.011 mg. of carotene per 100 gm. in March milk and 0.035 mg. in September market milk. From these and other studies it is evident that the three ice creams used in this study were a much richer source of carotene than is whole milk. It is generally

believed that fresh vegetables, particularly the green leafy vegetables, are especially rich sources of carotene for the human dietary. In their determination of pure carotene in green vegetables, Fraps, Meinke and Kemmerer[10] found that green string beans contained 0.03 mg. of carotene per 100 gm., beet tops 3.2 mg., sweet potatoes 4.5 to 5.7 mg., and carrots 4.5 to 8.4 mg. Zimmerman, Tressler and Maynard[11] report from a study of carotene in fresh and frozen vegetables that bush lima beans contain 0.24 mg. per 100 gm., spinach 0.46 mg., asparagus 0.70 mg., and broccoli 1.0 mg. Unfortunately, there is likely to be a serious loss of carotene from commercial vegetables after they are harvested, particularly if they are exposed to summer temperatures in stores or on roadside stands for a considerable period of time.

Since milk is generally considered to be a rich source of riboflavin, it is interesting to compare it and the ice cream under discussion. In studies conducted on milk produced by different breeds of cows in various stages of lactation and fed a variety of rations, Snell and Strong,[12] Hand and Sharp,[13] Houston, Kon and Thompson,[14] Johnson, Maynard and Loosli,[15] Holmes, Jones, Wertz, Esselen and McKey[16] and other investigators found that the riboflavin content of milk varied from 0.06 to 0.34 mg. per 100 gm., with an average value of less than 0.20 mg. Thus, on a weight basis, the ice cream under consideration is a richer source of riboflavin than is average milk, but ice cream is sold on a volume basis, — that is, by the pint or quart, — and because of the large amount of air that is incorporated, it contains much less riboflavin than does an equal volume of milk.

Eggs are considered one of the richest food sources of riboflavin. Engel, Phillips and Halpin[17] found from 0.064 mg. to 0.700 mg. of riboflavin per 100 gm. of albumin, depending on the riboflavin content of the hens' ration. Norris and Bauernfeind[18] also found that the ration influenced the amount of riboflavin in eggs, for fresh eggs from six flocks fed egg mash contained 0.207 mg. per 100 gm., whereas those from five flocks fed breeder mash contained 0.247 mg. Snell and Quarles[19] report that whole, white leghorn eggs contain 0.200 mg. of riboflavin per 100 gm. It may be concluded from these and other studies that commercial whole eggs contain about 0.20 mg. of riboflavin per 100 gm., and thus are not so rich in this vitamin as is the ice cream under discussion.

According to Cheldelin and Williams[20] such common foods as beef round, pork loin, veal chop, leg of lamb, mutton shoulder, chicken leg and oysters contain about the same amount of riboflavin as was found in the ice cream under discussion, but these authors found much less riboflavin in whole-wheat bread, chicken breast, salmon, dried lima beans, cabbage and carrots, which are widely used foods. Hodson[21] studied the riboflavin content of about fifty fruits and vegetables. Of these only one, broccoli, approached ice cream in riboflavin value, and only six or eight of the others contained half as much riboflavin. Thus, one may conclude from this and other studies that the ice cream under consideration compared favorably with practically all other foods as a source of riboflavin for the human dietary.

Summary

It has been estimated that the retail value of the ice cream consumed in this country annually is about one fourth that of milk and over twice that of eggs, apples or potatoes.

Samples of coffee, maple and vanilla ice cream, manufactured under plant conditions were assayed for carotene, riboflavin and ascorbic acid. The coffee, maple and vanilla ice cream contained 0.09 mg., 0.12 mg. and 0.09 mg. of carotene and 0.27 mg., 0.26 mg. and 0.26 mg. of riboflavin per 100 gm., respectively. No ascorbic acid was found, probably because of the large amount of air incorporated in commercial ice cream to increase its bulk.

Comparison of these ice creams with numerous widely used foods shows them to be excellent sources of carotene and riboflavin for the human dietary. The nutritive value of ice cream, however, varies with the nature and amount of the ingredients used and the process of manufacture.

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CLINICAL NOTE

SPOROTRICHOSIS*

Report of a Case

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SPOROTRICHOSIS is seldom seen in the United States and is easily confused with a number of other diseases. Strictly speaking, it presents a dermatologic problem, but the surgeon may encounter the disease in its early stages when it resembles a local infection. As it progresses and lymphatic invasion occurs, it becomes evident that one is dealing with a granuloma. For this reason, one should recall the possibility of sporotrichosis when considering a skin lesion that may be due to syphilis, tuberculosis, pyoderma, coccidiomycosis, actinomycosis or tularemia.

Sporotrichosis was first described as a pathogenic entity by Schenck in 1898. Since then, about 200 cases have been reported in the United States. Five of these were from New York State, and most of the others were from the Mississippi Valley.

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The responsible organism is *Sporotrichum schenckii*, a fungus probably acquired in this country through contact with a plant, such as the barberry shrub, a carnation or sphagnum moss. A break in the skin seems necessary as a portal of entry, since in most of the reported cases there is a history of an open lesion while the patient was in contact with the plant. The incubation period ranges from three days to three weeks. The primary lesion appears as a deep pustule at the site of entry, which may either become chancroid in character, with induration, softening and abscess formation, or take the form of an indolent ulcer. Later the regional lymph channels become involved, causing a lymphangitis, with nodulations along the channels simulating enlarged lymph nodes, but the nodes themselves are not involved. The skin surrounding these nodules is usually reddish purple. The nodules frequently break down, appearing like the original lesion. This is the most frequent form of the disease; it is described in textbooks as the "localized lymphangitic type." Constitutional symptoms are rare, and the lesions are usually painless. Some authors believe that surgery is contraindicated, but others have shown that scar formation can be greatly reduced by open drainage.

Figure 1. *Photograph of the Primary Lesion.*
This occurred on the inner aspect of the thumb, with nodulations extending upward on forearm.

There are four other types of the disease—disseminating subcutaneous, disseminating ulcerative, epidermal and systemic. The disseminating subcutaneous type is the same as the local one except

that the lesions are scattered over the entire body without following any pathway, suggesting an invasion of the blood stream. The disseminating ulcerating type is likewise the same except for much earlier ulceration. The epidermal type involves only the skin, but if these local lesions remain untreated, the deeper structures may become involved. The systemic type affects the bones, joints and muscles and is becoming more widely recognized as the causative factor in many obscure pulmonary lesions. In this phase the outlook is serious, but the localized lesions readily respond to potassium iodide therapy.

The fungus is extremely difficult to find by direct smear, but Lawless suggests, as a quick method of diagnosis while awaiting the result of cultures, adding a drop of Unna-Pappenheim's methyl green-pyronin stain to a drop of pus, after which the fungus can frequently be found by direct examination. Culture is the only definite method of diagnosis and is usually reliable. Pathological examination of tissue is not helpful, since the histologic picture resembles that of all the granulomas. Agglutination and cutaneous tests are of equivocal value.

Case Report

A 41-year-old captain was admitted to the station hospital on December 2, 1944, complaining of an infection of the left thumb, with "streaks and lumps" running up the arm. He had been on a course of sulfadiazine and 100,000 units of penicillin, as well as hot compresses to the thumb. Prior to admission the lesion on the thumb had been incised. At no time had the patient felt ill or had a fever, and except for the local lesions he appeared well. On the inner aspect of the left thumb toward the flexor surface there were several deep pustules about the size of the head of a match. Around this area the skin was thin and blue (Fig. 1). Along the volar surface of the forearm were many slightly tender nodules that felt like enlarged lymph nodes. There was no involvement of the axillary nodes. The temperature, pulse and respirations were normal and remained so throughout hospitalization. Urinalysis and a Wassermann test were negative, The white-cell count was 7200 with 79 per cent neutrophils, 18 per cent lymphocytes, 1 per cent mononuclear cells and 2 per cent basophils. The red-cell count was 4,750,000, and the hemoglobin 15.5 gm.

One of the pustules was opened, and a thin milky fluid exuded, a stained smear of which was negative for ordinary bacteria. Further questioning brought out the fact that 5 days before the appearance of the primary lesion the patient had been hunting rabbits, which suggested tularemia. At that time the lesions took on the characteristics of a granuloma: the nodules began to break down, and the surrounding skin grew reddish purple, with slight red streaking along the lymphatics (Fig. 2). One of the suppurative nodules was opened for culture, and the surrounding skin was removed for pathologic study, which showed only granulomatous inflammation of nonspecific character. All the direct stained smears were negative for organisms. Agglutination tests for tularemia were negative, as were guinea-pig inoculations, but after 10 days the cultures on dextrose agar yielded *Sp. schenckii* (Fig. 3). On closer questioning the patient stated that he had been trimming barberry shrubs at his home in Pennsylvania 2 weeks before the lesions appeared.

When the diagnosis had been made, the patient was given a saturated solution of potassium iodide, with a starting dosage of 10 drops three times daily, which was gradually increased to 40 drops three times daily and was maintained at that level. There was marked regression of the lesions; they had entirely disappeared 4 weeks after the commencement of medication, but the iodide was continued for 4 weeks longer to prevent recurrence of the fungus.

Figure 2. *Photograph of Lesions at a Later Stage.*
The nodulations ran along the volar aspect of the arm and showed characteristic ulceration and suppuration. They involved only the distal third of the forearm.

Summary

A case of sporotrichosis is reported, primarily for its surgical interest, since in the early stages the disease often presents itself as an ordinary infection.

Early diagnosis is important, since treatment with potassium iodide is specific and prevents further advance of the disease.

The diagnosis is made through cultures, but it must be remembered that the cultures require five to seven days before showing growth.

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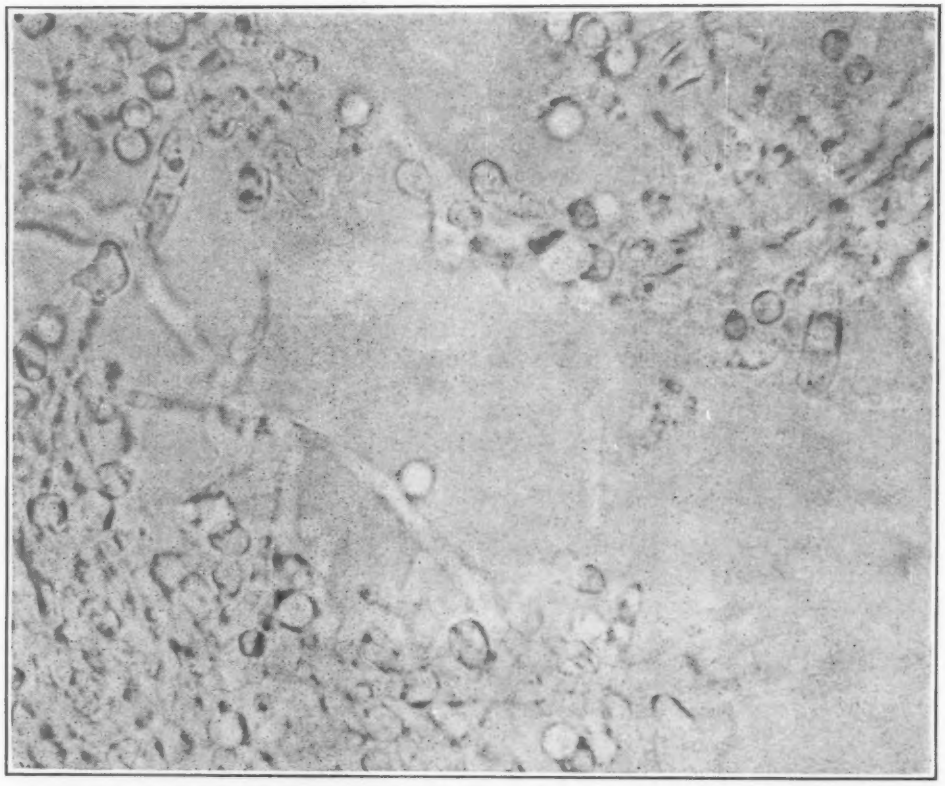

FIGURE 3. *Hanging-Drop Preparation Showing Characteristic Morphology of Sporotrichum (high-power).*

SYMPOSIUM ON MEDICAL SOCIOLOGY

PROBLEMS IN THE DISTRIBUTION OF MEDICAL CARE*

DEAN A. CLARK, M.D.†

IT IS no accident that the Harvard Medical School has decided to hold these lectures on medical sociology. Never before has there been such widespread interest and activity in the social aspects of medicine among all groups of people — not only medical students and practicing physicians but farmers, members of labor organizations, businessmen and government officials. Ten years ago it would not have occurred to anyone to have such a lecture series, but today it seems the most natural thing imaginable.

Indeed, the question of how better to distribute medical service is constantly present. Among groups of physicians, for example, the New York Academy of Medicine has been holding a series of fifty or more weekly meetings devoted to "Medicine and the Changing Order." Then too, one can scarcely pick up an issue of a newspaper — the *New York Times*, for instance — without finding an article or perhaps an editorial dealing with the subject. Only last month a major article in *Fortune* was devoted to it, and radio forums touch on it nearly every week.

One is also reminded of the general interest in these matters by the polls of public opinion that have been appearing with increasing frequency. Although these polls differ considerably from one another in detail, all without exception show that the majority of the people believe that some change is necessary in the ways in which medical care is being distributed. A poll conducted by the American Medical Association among medical officers of the Army and Navy indicated that most of those who replied were unwilling to return to the individual practice of medicine on a fee-for-service basis.

Another constant problem is the fact that some 12,000,000 Americans now serving in the armed forces will, as veterans, have enduring rights to hospital care for both service-connected and non-service-connected disabilities for the rest of their lives, at the expense of the federal government. It is difficult to say, moreover, just what the effect will be on the millions of soldiers and sailors — and their thousands of physicians — of having received or furnished comprehensive medical service of the highest character for three years or so without the payment of any fees at all.

Besides the purely military medical services, the war has brought a number of new activities in medicine affecting civilians, and these too are bound to affect the future of medical practice in this country. Think, for example, of the aid given to medical students by the Army and Navy. For the first time, men who wished to study medicine have been enabled through this aid to do so without regard to their financial status. Similar opportunities in nursing have been offered by the Cadet Nurse Corps. In the realm of research, the Office of Scientific Research Development, through its Committee on Medical Research, has financed an incredible amount of important scientific work in medical schools, hospitals and laboratories. The Emergency Maternal and Infant Care Program supervised by the United States Children's Bureau has paid, with federal funds granted to the states, for the care of the wives and infants of hundreds of thousands of enlisted men. It is impossible to believe that some of the benefits of these wartime activities will not be continued in one form or another after the war is over.

Other indications of the desire for improved distribution of the benefits of modern medicine are to be found in the public actions of various influential persons and groups. Less than a month ago, for example, Governor Warren, of California, stated his intention of supporting compulsory health insurance in that state. Mayor La Guardia, of New York City, has announced the initiation of a plan to provide 1,000,000 residents of that city with medical care on a prepayment group-practice basis. The American Public Health Association has recently gone on record as favoring a nationwide system of medical care for all people, supported by compulsory insurance, general taxation or a combination of the two. In another statement, some thirty physicians and laymen have urged national action to furnish complete medical care of high quality to the American people. Included in the group, incidentally, was a physician who is a recent president of the American Hospital Association and is now chairman of the Commission on Medical Care appointed by Governor Dewey, of New York. Another physician-signer of the statement has recently served two terms as president of the Massachusetts Medical Society.

Finally, of course, there are the prospects of official governmental action in this and other countries. Here there is the much discussed Wagner–Murray–Dingell Bill; in Great Britain there is the Churchill government's "White Paper" proposing a national health service; in Canada, several years of study have yielded a national health bill that includes compulsory insurance, which has been approved in principle by the Canadian Medical

*This is the second of a series of nine lectures on medical sociology given weekly at Harvard Medical School during January, February and March, 1945; they were sponsored by the Department of Preventive Medicine and were primarily intended for third-year students. These articles will temporarily replace the reports "Medical Progress."

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Association. All these proposals are sweeping in character, and they indicate the trend among important sectors of legislative opinion.

So there can be little doubt that this series of lectures is indeed timely. All this interest in the better distribution of medical and health services is certainly noteworthy, but one may well ask why it has come about. Is it purely accidental that so many people are concerned about this subject, or has the feeling been aroused by a small, highly vocal minority? Or are there, perhaps, deeper reasons for it? Perhaps the title of this lecture should be changed from "Problems in the Distribution of Medical Care" to "Is There a Problem in the Distribution of Medical Care?" with the subtitle, "If So, What Is the Nature of the Problem?"

First, is there really any problem? With the lowest over-all national death rate in its history and one of the lowest in the world, should not the United States be perfectly content to let well enough alone? This is a point of view one often hears expressed. But the over-all death rate is a sweeping average and, as every science student knows, one must look behind an average to see what makes it up. Here are a few examples of disparities in death rates.

In 1941, when the national death rate was 10.3 per 1000 for Whites, it was 13.7 for Negroes. In 1942, the death rate for tuberculosis for all people was 42.2 per 100,000. For Whites alone in that year it was 34, but for Negroes, it was 112, and for Indians 230. When the death rate for infants under one year of age — often considered the most accurate index of health — was 45 per 1000 live births for all people in 1941, it was only 31 in Connecticut, but in New Mexico it was 95.

It is easy to see from these examples that over-all death rates do not tell the whole story. But it may further be asked what information even the most refined figures on death rates give about people's health and their need for medical care. Obviously, figures on deaths tell practically nothing about the state of positive health, since they give no indication how many of the population are really well nourished, full of physical vigor and emotionally well adjusted. What is more, death rates do not even reveal much about the prevalence and duration of disease and disability and therefore about the need for medical care.

Indeed, a lower death rate may well be accompanied by a higher rate of disability, paradoxical as this may sound. For instance, much of the dramatic fall in the national death rate in this country has been due to control of communicable disease, especially diseases of childhood. As everyone knows, people live longer and there are proportionately more elderly persons than there used to be. The population is aging. But of course elderly persons are far more susceptible than are young ones to chronic diseases such as arthritis, cardiovascular-renal disease and diabetes, which result in much longer disability than do most diseases of childhood. As another example, a few years ago a man might come down with an overwhelming lobar pneumonia and be dead in three days in spite of the best treatment available. Today, with the sulfonamide drugs and penicillin, such a patient might well be cured, but he would be disabled for at least thirty days — ten times the disability, ten times the need for medical and nursing care, but a live patient and a decreased death rate.

Such figures on the incidence and duration of disease and disability as exist cannot be compared with those of a generation ago, because at that time such surveys had not been made. But the examples given above at least make it clear that lowered death rates do not tell much about the state of the Nation's health and do not imply that there is less disability or less need for medical care than there was in earlier years. It seems quite probable, in fact, that the country needs more medical care than ever before, but the kind required is somewhat different. There is relatively less need in medical practice for immediate and dramatic life-saving measures; there is need for more preventive service, more care of chronic illness and more attention to the emotional, psychologic and social aspects of illness. This changing pattern of medical practice is not always fully realized — or liked — by physicians, but there is no doubt that it is taking place.

It therefore seems reasonable to conclude that there is indeed a problem in the distribution of medical care. What, then, is the nature of the problem? Is it primarily a lack of adequate scientific knowledge? Obviously, this need exists and must be met, but no one can look at the present situation and not agree, also, that the knowledge now at hand is not being used effectively. The venereal diseases, for instance, can be wiped out today, but the country is still, in spite of much excellent work, a long way from this goal. So that the present problem in medicine is not simply a need for more knowledge.

Is the need principally one for more and better facilities and trained personnel? Of course this lack does exist, but in the case of physicians, for instance, once they have been released from the armed forces the Nation will not be so badly off so far as sheer numbers are concerned. The real question is whether they are available to the people who require their services. Much the same thing is true of hospital beds and trained nurses. Certainly more of them are needed, but one must also stop and ask whether the best use is being made of those now at hand. In some fields, it is true that there are absolute numerical shortages. For instance, there are not nearly enough dentists, public-health nurses, psychiatrists or medical and psychiatric social workers. Even with these groups, using what there is to the best advantage would make a

vast difference. In short, lack of facilities and trained personnel is by no means the heart of the problem.

Is the problem perhaps lack of money? The people of the United States are spending more than $4,000,000,000 a year for health services, including all funds spent by private patients, industry, charities and foundations and government agencies. To be sure, more money could be used advantageously, but many health and medical services are already being supported with great generosity. In fact, if all the funds now expended were used more systematically and distributed more equitably, the present amount would not be far short of adequate. Lack of funds is not, therefore, the entire basis of the difficulty.

If the weaknesses in the present distribution of health and medical services are not due solely to lack of scientific knowledge, facilities, trained personnel or funds, what is the real nature of the problem? There are several ways of describing it — or, it might be said, several elements in it. First, because it is perhaps the element recently most talked about, let us consider the inequitable distribution of personnel and facilities. Before the war there was one physician to 750 people in the United States, but in New York State there was one to 500 people, whereas in Mississippi there was one to 1500. Similarly with general hospital beds, there was one to 250 persons for the country as a whole, with one to 200 in New York State and one to 650 in Mississippi. And, it should be said, differences between communities within each state were as great as those between states. Even greater inequities exist in the distribution of nurses and dentists.

There can be little doubt that this uneven distribution is fundamentally the result of general economic conditions. In 1938, for example, counties with an annual per capita income of more than $600 had eight times as many physicians relative to the population as did counties with one of less than $100. Another factor is that physicians do not want to practice in rural areas. In 1938, even the wealthiest group of rural counties had 30 per cent fewer physicians relative to the population than did urban areas of the same per capita income. A third factor is the presence or absence of hospital and diagnostic facilities. Among counties with an annual per capita income of $300 or less, those with no general-hospital beds had 60 per cent fewer physicians relative to the population, than did those with reasonably adequate general-hospital facilities. During the war, the inequities of distribution have grown worse. The result is that by the end of 1943 almost one third of all the counties in the United States had more than 3000 persons per physician.

The situation has been summed up in another way by Davis[1]:

> Communities of under 5000 population had 48 per cent of the population of the United States in 1929, but only 30 per cent of the physicians, and these physicians received only 18 per cent of the total gross income of all privately practicing physicians. Cities of between 5000 and 100,000 population had 23 per cent of the people and 26 per cent of the physicians, and these physicians obtained 28 per cent of the total physicians' income.
>
> Cities of 100,000 and over had 29 per cent of the population and 44 per cent of the physicians, and these physicians received 54 per cent of the total gross income.

Another major element of the problem is the present pattern of payment for medical care. The provision of such care may be said to depend on two factors: medical need and medical demand. Medical need is composed of the actual physical and emotional requirements of a patient for a physician or for other services. This need can, of course, be determined only professionally — that is, by a physician through a medical history, a physical examination, laboratory tests and so on. But medical demand is quite another matter, and it does not necessarily correspond at all with the medical need. This demand is composed of at least three elements. The first of these is the psychologic and educational background of the patient. Is he afraid of all doctors or is he perhaps overanxious to use them? Does he know how and when to make use of available medical services? The second element is the physical presence or absence of medical personnel and facilities. There is always less demand on physicians or hospitals by patients who have to travel far to reach them than by those nearer at hand. The third element is the economic status of the patient. Can he afford to pay the physician's or hospital's bill, or is he afraid he may be unable to do so and therefore hesitant to seek medical aid? Demand is affected, too, by whether the method of payment is that of fees for service, which acts in many cases as a barrier to obtaining service, or by periodic prepayment or insurance, which requires no further payment at the time of receiving service and therefore usually increases medical demand.

It is of course medical demand that largely determines the distribution of medical personnel and facilities and the receipt of medical care. It should include general social, educational and economic conditions, as well as physiological needs. These conditions are clearly beyond the power of the medical profession alone to change. The conclusion inescapably follows that the profession alone cannot appreciably alter medical demand and therefore cannot hope to influence or affect greatly the distribution of medical care. Since the latter ultimately depends on general economic and social factors, it clearly can be much altered only by general public action. The medical profession can, however, play an important role in assisting and guiding such public action into constructive and fruitful channels.

The principal facts concerning payment for and receipt of medical care today may be briefly summarized as follows. Families with incomes of less than $1200 a year pay on the average $43 a year per family for medical care. The same group annually receives 1900 physicians' house, office and clinic calls per 1000 persons. Forty-seven per cent of such persons receive no medical care in any one year, but this group has sixteen days of disability per capita annually. Those earning from $2000 to $3000 pay $90 a year per family for medical care and receive annually 2300 physicians' calls per 1000 persons. Only 37 per cent receive no medical attention in a year, and the group has only six days of disability per person per year. The group with incomes of $10,000 pays $500 a year per family for medical care and receives annually 4700 physicians' calls per 1000 persons. Only 14 per cent receive no care each year, and annual days of disability are less than six per person.

It thus appeared that the lower the income group, the more days of disability and the fewer physicians' calls. The heart of the major problem is illustrated by these facts. In general, it is safe to say that, except for days of hospital care in urban centers with well developed public-hospital systems, the least care gets to the people who need it most, that is, those who have the most illnesses and the most days of disability but the least money to pay for medical care. Medical demand certainly does not follow medical need.

The problem of payment for medical care divides into two parts. First, there is what might be called "absolute inability" to pay the full cost of care, whatever the method of payment. Among families with incomes below a certain level, say $1200 a year, the costs of medical care of reasonably good quality obviously cannot be met. Even in the prosperous year 1943, about one third of the country's population fell into this group. Persons in the lower economic levels also have larger families and more sickness than have the well-to-do. This combination of circumstances makes it totally impossible for these people to pay the full costs by themselves, even on a prepayment basis, and what they can pay must be supplemented from other sources.

It is sometimes suggested that people might be induced by educational measures to increase the amount they would be willing to spend for medical care. The proportion of family income expended for automobiles, washing machines, moving pictures, cosmetics and so forth has certainly come up sharply in recent years. Why not the same thing for health services? Possibly it could be done, but health expenditures have to compete with other human wants. Besides, people buy automobiles, lipsticks and washing machines because they *want* them — and nobody wants to have to see a doctor or a dentist.

In addition to this absolute inability to pay the full costs of medical care, there is the even more important second part of the problem of payment — namely, relative inability to pay. This affects not merely families at the bottom levels of income but those of all economic levels except perhaps the top 5 per cent. This problem arises from the fact that the incidence and severity of illness are unpredictable for the individual, yet, under the fee-for-service system, he is obliged to pay only when illness occurs. Thus, with the traditional method of payment the heaviest burden falls, in any one year, on the few persons unfortunate enough to be ill, and this burden strikes just at the time when, for the wage-earner, his income is cut off — namely, when he is ill.

For instance, 10 per cent of all the families in the population pay in a given year 41 per cent of the costs of medical care for the whole population and thirty-two per cent pay 41 per cent, so that the remaining 58 per cent pay in any one year only 18 per cent of the total cost of medical care for the country. The incidence of illness is uneven and consequently the distribution of the costs of medical care is also uneven under the fee-for-service system. Many a family with the absolute ability to pay $150 or more a year for medical care under a planned periodic-prepayment system can suffer serious hardship or even be crushed financially by the enormous costs of a single serious illness, billed all at one time.

Two elements of the problem have so far been considered — the difficulties of the distribution of personnel and facilities and those of payment for service. But what about the organization of medical services? Medicine is no longer a one-man science: it takes diverse skills, expensive equipment and much trained assisting personnel to do a decent job today.

Now, typically, — and it *is* still typical, — doctors are in individual practice, with considerable duplication and waste of resources. Each has an office, the necessary equipment for x-ray examinations, metabolism tests, electrocardiograms, and laboratory procedures and an office nurse, technician or secretary. He cannot possibly use all these to their full capacity. For example, a friend of mine practicing in a Southern city is proud of his teaching appointments at two medical schools. He also has a private office, with an x-ray machine, metabolic and electrocardiographic apparatus, an adequate laboratory and an office nurse-technician-secretary. Because of his teaching and research and home and hospital calls, he is in his office only two hours a day, three days a week. All the rest of the time his fine equipment and office assistant are virtually idle.

Other evidence of poor organization in the medical services of today is found in the obvious waste of professional skill. Another friend of mine while

a resident in surgery at one of the best teaching hospitals performed one hundred and fifty major operations, most of them with little supervision. His first year in private practice, in a town where he was immediately given a hospital staff appointment and was welcomed by the profession, he did only two major operations. This is obviously a serious waste. After a number of years, he may be expected to increase his work to near his capacity, but think of the wasted skill in the intervening years. Also, before he reached this point he may lose much of the technical perfection that was probably at its peak at the close of his year of residency.

Just as important a result of the present inadequate organization of medical services as its waste of resources and professional training is its failure to assure, for the benefit of the patient, the wide diversity of skills essential in good, modern medicine. The physician in individual private practice has to exercise all his ingenuity if he is to obtain for his patients — especially for the less wealthy ones — all the special services that they require. In the first place, specialists and laboratories may simply be unavailable, as in many small towns and rural areas. Even in large cities, general and special physicians and facilities are not related in an orderly manner, and the practitioner may have great difficulty in finding exactly the service that his patient needs. Second, the physician may hesitate to refer patients for consultation or laboratory tests because he knows that they will have to pay for these and it may be hard for them to do so. He may even begin to lose patients if he is too free in advising special work; he begins to get a reputation for "always sending you to a specialist," and patients hesitate to go to him for fear of the added cost. Then, too, patients who go to a specialist sometimes do not return to the original physician at all. For all these reasons, many a physician tries to handle far more than he is competent to, or he may be tempted by offers of split fees from certain specialists. In either event, the patient is the sufferer — he does not receive specialist care solely because of his medical need for it.

The lack of organization is also illustrated by competition rather than co-operation among hospitals. Take the example of four competing hospitals in a small town or in four small towns near one another — a not infrequent occurrence. Each small hospital is trying valiantly, pitifully, to do everything, and of course is unable to do so satisfactorily. A single large hospital could usually provide far better facilities and equipment than any one of the four. Yet consolidation of such rivals is all too often the exception rather than the rule. Planning of hospitals by areas or regions and by special tasks that each can be expected to perform has been strongly advocated for years, but only recently has any serious attempt been made to carry out such plans.

There have been discussed three elements of the problem of distribution of medical care: the distribution of personnel and facilities, payment for care and organization of services. A fourth element is especially important to a group of medical students — namely, the position of the physician, and the way in which these matters affect his professional and economic opportunities.

This should first be examined from the professional outlook. Medical students are trained in every medical school and teaching hospital in this country under a system of co-operative group medical practice. They learn to apply, for the benefit of their patients, the best available technics and to use the best equipment, with the generous assistance and consultation of their ablest colleagues and teachers. Yet they usually go out into individual private practice, where they are simply unable, even with the best will in the world, to practice the kind of medicine that they learned in their training or, in many cases, to advance their professional skills while they are practicing. The practitioner is "on his own," indeed, and to be so often forces him to practice progressively poorer medicine.

Fortune[2] puts it thus: "Many doctors, on going from the group practice of their internship to a largely solo method of practice, have felt that they have dropped back to a geologically older level of medicine on which they cannot hope to realize their maximum potential for service."

One may think that the professional frustration suffered by large numbers of physicians is compensated for, to a large extent, by their economic rewards, but such is not the case. It is true that few doctors starve, but fewer still get rich. All have necessarily a serious economic problem to face. To begin with, at least $10,000 or $12,000 has been spent on the medical education of each. The average net income of all physicians in 1929 was $5300 a year. By 1934, the average had dropped to $3300, and by 1941 it had come back to about $5000. The average net income is certainly higher during the war, but in general the peacetime figures are indicative of the economic status of physicians. The average, however, does not tell the whole story in this instance either. Two thirds of physicians had incomes less than the average — which of course means that a few had extremely large incomes to bring up the average. Half of all physicians in practice were actually earning less than $4000 net a year in 1929. This is none too much to repay the costs of medical education and to provide for a family and for retirement after sixty-five — few physicians earn much of a livelihood after that age. For every physician who had a net income of $10,000 or over in 1929, there were two with incomes of less than $2500.

In general, the physicans with the poorest economic status are young practitioners, general practitioners of any age and rural practitioners of any type and age. The best off are specialists in medium-sized cities who have been seventeen years in practice. It is no wonder that doctors flock into the specialties and into cities. Who would not? One cannot blame them for doing this, because they all have to try to reach a high earning power for at least a few years. The trouble is, of course, that many do not succeed. Statistically speaking, only one in eight of this class will ever have a net income of over $10,000 a year. Naturally, each is sure that he will be the one. But it is difficult to predict.

Clearly, it is not the physician's fault that his basic economic insecurity obliges him to have a constant and intense interest in his income, to practice where money is and to keep up in the competitive race, sometimes at the expense of his professional skill and integrity. But, regardless of where the fault lies, the fact remains that physicians have a far from satisfactory economic position in the present scheme of things, and their economic position has unfortunate effects on medical practice.

* * *

In summary, there is a problem in the distribution of medical care; the problem is not just a need for more scientific knowledge, facilities, trained personnel or money, however much some or all of these are needed. It has a number of aspects. There is inefficient and inequitable distribution of medical personnel and facilities; present methods of payment for care result in the fact that those with the greatest medical needs, such as the low-income groups, the aged and so forth receive the least care; a large section of the population could not afford to pay the full cost of all the care they require by any method of payment; another large section cannot pay its full costs without hardship because the incidence and severity of illness are unpredictable, yet under fee-for-service payment the costs must largely be met at the time sickness occurs; there is great economic and professional waste in the present inadequate organization of medical services; the diverse skills and expensive equipment of scientific medicine are poorly integrated to give the best service to the patient; and finally, many physicians have little prospect of either professional satisfaction or economic security in the practice of medicine.

These facts present a serious challenge. There is truly a crisis in American medicine today. In the lectures to come you will hear more about these problems and about the attempts being made, here and abroad, to solve them. Certainly, the open, frank and dispassionate discussion of problems is the healthy means of reaching their solution. This you will have in this lecture series.

There is one more thing to remember. The problems of the distribution of medical and health services are not confined to the medical profession, nor can they be solved by it alone. Health is not the property of physicians but of everyone. These problems are the problems of all the people. To solve them therefore means discussion and action not only by physicians but by the medical profession as a whole, together with those in business, agriculture, labor and government, if we are to look forward with assurance to better health for the American people.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 32021

PRESENTATION OF CASE

First admission. A twenty-five-year-old man entered the hospital because albumin had been found in the urine fifteen months previously. He had had no complaints, except for occasional pain over the costovertebral angles.

Physical examination was negative.

The temperature was 98.6°F., the pulse 75, and the respirations 20. The blood pressure was 98 systolic, 78 diastolic.

The red-cell count was 4,460,000, with 80 per cent hemoglobin. The white-cell count was 7400, with 60 per cent neutrophils. The urine was cloudy, yellow and alkaline, and had a specific gravity of 1.014. It gave a ++ test for albumin; the sediment contained 3 red cells, 20 white cells and 2 epithelial cells per high-power field, and amorphous phosphates, triple phosphates and a few bacteria were seen. The nonprotein nitrogen was 20 mg. per 100 cc., the serum calcium 10.4 mg., and the phosphorus 3.2 mg. A blood Hinton test was negative. An intravenous pyelogram showed a small area of calcification in the region of the middle calyx of the right kidney; the dye was excreted a little better by the left than by the right kidney. The middle calyx of the right kidney was deformed, and the area of calcification described above was included in the dye. Although both ureters were rather large, there was no definite evidence of hydronephrosis. The urinary bladder was not remarkable.

Cystoscopy showed a slightly reddened bladder. Both ureteral orifices were normal, and catheters were passed to both kidneys without difficulty. A normal flow of slightly hazy urine was obtained from each kidney. Intravenous phenolsulfonephthalein appeared in three minutes from the left kidney and in four minutes from the right. The urine from each kidney showed no cells or organisms and was sterile on culture. The patient was discharged three days after admission.

Second admission (five years later). During the first three years after discharge the patient had three brief episodes of severe pain in the right flank, which radiated to the right lower quadrant. At the end of that time, several stones were removed from the bladder at another hospital. After this operation he felt well until three months before readmission, when he noticed that he tired easily. He had a sacroiliac ache on exertion, which subsided on rest. He had lost no weight. Three weeks before readmission he passed a stone by urethra.

Physical examination was negative.

The temperature was 98.6°F., the pulse 90, and the respirations 20. The blood pressure was 100 systolic, 60 diastolic.

The hemoglobin was 13.8 gm. The white-cell count was 7900. The urine was cloudy, amber and acid, with a specific gravity of 1.018. It gave a + test for albumin. The urinary sediment contained a rare red cell, 10 white cells and a rare granular cast per high-power field. Culture of the urine revealed on one occasion *Staphylococcus albus*, and on another, colon bacilli and nonhemolytic streptococci. The total phenolsulfonephthalein output was 45 per cent in two hours. The nonprotein nitrogen was 30 mg. per 100 cc., the serum protein 5.5 gm., the calcium 10.4 mg., and the phosphorus 3.0 mg. The chloride was 102.1 milliequiv. per liter, and the carbon dioxide combining power 26.3 milliequiv. The uric acid was 4.4 mg. per 100 cc. An x-ray film of the teeth showed no evidence of apical abscesses or retained roots. An intravenous pyelogram revealed several areas of calcification scattered over both kidney shadows. Intravenous dye appeared promptly in good concentration. There was some distortion of the calyxes on both sides, more marked on the left than on the right. The right calyxes, pelvis and ureter were dilated, but a definite stone was not visible within the lower ureter.

The patient was discharged improved on the tenth hospital day.

Third admission (one year later). During the year after discharge the patient complained of intermittent, dull, burning pain over both costovertebral angles, which usually appeared in the morning following urination and lasted several hours. He passed several stones. He complained of no frequency, dysuria or bone or joint pain. The serum calcium had remained at about 10.4 mg. per 100 cc., and the phosphorus at 3.0 mg. On one occasion, after ten days on a low-calcium diet, the urinary calcium output was 282 mg. in twenty-four hours.

Physical examination revealed circumscribed, papular, scaly, erythematous lesions, 1 to 4 cm. in diameter, on the inner aspect of the groins, the right lower leg and the forearms.

The temperature was 97°F., the pulse 90, and the respirations 25. The blood pressure was 110 systolic, 70 diastolic.

The hemoglobin was 14.9 gm. The white-cell count was 8100. The urine was light amber and acid, with a specific gravity of 1.010. It contained 3 red cells, 10 white cells and a rare hyaline cast per high-

power field. A urinary culture showed abundant colonies of *Staph. albus* on one occasion and was sterile on another. A phenolsulfonephthalein test showed 47 per cent excretion in two hours. The nonprotein nitrogen was 30 mg. per 100 cc., the total serum protein 7.3 gm., the calcium 10.6 mg., and the phosphorus 2.7 mg. The chloride was 102 milliequiv. per liter, and the phosphatase was 6.1 Bodansky units per 100 cc.

Early on the fifth hospital day the patient began to complain of generalized aches and pains, headache and an infrequent, nonproductive cough. During the day, malaise and cough continued and discomfort developed over the sternum and the left upper chest anteriorly. There was some tenderness to percussion over the left anterior chest just below the clavicle. The breath sounds were questionably diminished over this area, without dullness or rales. The abdomen was normal. An x-ray film of the chest was negative. Hemolytic streptococci were cultured from the sputum. The evidences of infection subsided in five days and the patient was discharged.

Fourth admission (two and a half years later). During the period after discharge the patient had had occasional episodes of cloudy urine; he had passed a stone about a year before admission. He complained of no chills, fever, hematuria or renal colic.

Physical examination was negative.

The temperature was 100°F., the pulse 90, and the respirations 20. The blood pressure was 108 systolic, 72 diastolic.

The hemoglobin was 14.3 gm. The white-cell count was 7800. The urinary sediment contained an occasional white cell and epithelial cell. The urine culture showed *Staph. albus*. The urinary calcium output was 328 mg. in twenty-four hours. A phenolsulphonephthalein test showed 60 per cent excretion in two hours. The nonprotein nitrogen was 31 mg. per 100 cc., the total serum protein 6.2 gm., the calcium 9.4 mg., and the phosphorus 2.6 mg. The phosphatase was 4.4 Bodansky units per 100 cc., and the chloride 102 milliequiv. per liter. In an x-ray film of the teeth the lamina dura was found to be preserved. A barium swallow showed that the esophagus and trachea were displaced slightly to the left in the upper neck. No definite defects were seen in the esophagus. There was no substernal displacement of the esophagus or trachea. There was a small soft-tissue thickening in the region of the thyroid gland. An intravenous pyelogram revealed many small flecky areas of calcification in the region of all the calyxes of both kidneys. All the bones showed slight decalcification. The kidneys were normal in shape, size and position. No unusual soft-tissue masses were seen in the abdomen. Intravenous dye appeared promptly in both kidneys; it showed no dilatation of the calyxes, pelvis and ureter on the left, but the right calyxes, pelvis and ureter were slightly dilated. The bladder shadow was not remarkable.

An operation was performed.

Differential Diagnosis

Dr. Joseph Aub: This is an "Albrightian" case, and I am sure that I am going to be educated.

Are there any x-ray films?

Dr. Benjamin Castleman: We have films, but there is no roentgenologist to interpret them.

Dr. Aub: There are flecks of calcium in the kidneys, which appear to be in the calyxes rather than in the parenchyma. The kidneys themselves do not look large. One can be sure that the calcium is in the calyxes because the areas of calcification are obscured by the pyelogram dye. There is some slight decalcification of bone.

We have, then, a man who had been going on for many years with repeated passage of kidney stones. The kind of stones is not mentioned, although I suspect that Dr. Albright knew whether they were phosphate, oxalate or uric acid stones.

Dr. Castleman: The record states that they showed a large amount of phosphate and a small amount of oxalate.

Dr. Aub: Since these were phosphate stones, it makes one think, of course, of parathyroid disease. This boy was carefully studied, however, over a long period of time and never had a high blood calcium and had only an occasional relatively low phosphorus. The high calcium output in the urine on two occasions is extremely suggestive of hyperparathyroidism and makes one think that that is the diagnosis. But that is not adequate evidence for me to make this diagnosis. I do not see the soft-tissue mass in the neck in the x-ray films, but it is said to have distorted the trachea and esophagus. But a parathyroid tumor is practically always so soft that it does not, in my experience, shift the esophagus. There is said to have been thickening around the thyroid gland, which is rather suggestive of a thyroid rather than parathyroid adenoma. In favor of hyperparathyroidism, however, are the high calcium output, the two low phosphorus determinations and the slight decalcification. This does not seem sufficient evidence to make a diagnosis of hyperparathyroidism in a patient who repeatedly had a normal blood calcium, often a normal blood phosphorus and a good lamina dura around the teeth, particularly when the disease had been going on for a long time, as judged by the renal calculi. On the contrary, it fits in better, it seems to me, with a renal condition described by Randall,* of Philadelphia, in which calcified areas appear beneath the mucous membrane in the calyxes of the kidneys and eventually break through, stones gradually accumulating around these denuded calcified areas. He found that the stones could be composed of

*Randall, A. Initiating lesions of renal calculus. *Surg., Gynec. & Obst.* 66: 201-208, 1937.

phosphate or oxalate or uric acid, depending on the deposition from the urine. With such a diagnosis, this patient would not be placed in the hyperparathyroid group, although I do not believe that the articles published by Randall rule out hyperparathyroidism in his cases.

I am willing to say that the surgeon who operated on this patient looked for a parathyroid tumor. I do not believe that he found it, nor do I believe that he even found large parathyroid glands, because I doubt that this man had enough renal damage to cause parathyroid hyperplasia. I think that he probably had an old pyelonephritis, which may have accentuated the production of stones.

Dr. Edward C. Reifenstein: I should like to give Dr. Aub some blood chemical determinations that I had in my office notes but that did not get into the hospital record.

Dr. Aub: One set of values is as follows: calcium, 11.2 mg.; phosphorus, 2.8 mg.; and phosphatase, 10 Bodansky units. Another is calcium 10.9 mg. and phosphorus 3.5 mg., with later a phosphatase of 5.3 units. Still another is calcium 10.3 mg., phosphorus 2.5 mg. and phosphatase 5.9 units. These are suggestive of hyperparathyroidism, I admit. I think that a search for overactivity of the parathyroid glands is justified, but if I had operated on this man, I should have looked for a parathyroid tumor without conviction, and I shall end up by saying that I do not believe that they found one.

Dr. Fuller Albright: If one gives a small dose of parathyroid hormone to a normal person, one may get increased calcium in the urine, although the blood calcium may still be normal. Thus, with a moderate degree of hyperparathyroidism, a patient can have a normal blood calcium and still have increased calcium in the urine and kidney stones; such a patient should have a low serum phosphorus, unless there is glomerular damage.

This case discussed by Dr. Aub is one of a large group of cases that we see in the Stone Clinic in which it is difficult to rule out a mild primary hyperparathyroidism. These patients show increased calcium in the urine, a low serum phosphorus and a normal or slightly low, occasionally slightly high, serum calcium. We think that these cases are due to pyelonephritis with resulting tubular damage, as a consequence of which the kidneys lose calcium far more readily than normal ones. The sequence of events in this group can be summarized as follows: pyelonephritis; a kidney that lets calcium through more readily than normal; a tendency to a low serum calcium; secondary hyperparathyroidism to meet the tendency to a low calcium; and a low serum phosphorus. One ends up with a normal serum calcium, a low serum phosphorus, a low calcium-phosphate ion product and, hence, a tendency to osteomalacia and a high serum phosphatase, which this man had. You can see that it may be difficult to differentiate this group of cases from those of primary hyperparathyroidism, which may have secondary urinary infection of the kidneys. As yet, we do not know any way of clinically differentiating these two groups of cases.

I do not believe that I disagree with Dr. Aub in anything except that I thought that a tumor would be found. We both agree that the patient should have been operated on.

Dr. Oliver Cope: In all these cases the surgeon has to have a certain sort of conviction that the patient has hyperparathyroidism, because if he is not convinced beforehand he may not go the whole length, and as you know, it is not always easy to uncover all four parathyroid glands. Obviously, uncovering three glands may not be enough under such circumstances, if the three uncovered turn out to be normal. If four glands are found with no gross evidence of disease, he must wonder if he has not overlooked a fifth, because a certain number of people have five glands. I emphasize this because the hunt often takes a long time. In other words, I believe the surgeon must be convinced that the diagnosis is correct.

I agreed with Dr. Albright, as opposed to Dr. Aub, that there was a good likelihood that this patient had hyperparathyroidism. So I was willing to take the time to look. The reason I say that is that I do not believe that Dr. Albright has enlarged enough on some of the experiences that we have had in operating on these patients. Only a year ago we operated on just such a patient. I went away from the table very discouraged, having uncovered four parathyroid glands that, so far as I could tell, were grossly normal. I took out the largest of the four because it was a little more globus in form than the other three, and to my great delight Dr. Castleman found a tiny, minute adenoma in it, and the subsequent changes of metabolism confirmed Dr. Castleman's anatomic diagnosis. In other words, what one must look for is not a big adenoma but rather something about the size of the head of a black-headed pin. To go back to the x-ray findings, I paid little heed to the report of the x-ray taken after the barium swallow. If there is displacement of the esophagus or the trachea or if there is a palpable nodule in the neck, it is a thyroid, not a parathyroid, tumor. Actually I could not find anything in this case to account for the x-ray findings.

At operation we uncovered four glands. They had a disturbingly normal color. The first one, the left lower, was a little larger than the average gland but the color appeared normal, that is, the relative distribution of fat cells to epithelial parathyroid cells seemed to be within the limits of normal. That was discouraging because, if there is a hyperactive adenoma in one, the other glands ought to be atrophic. This gland did not appear to be atrophic. The upper one on the left and the two on the opposite side were then uncovered, and all were within the normal limits. I took out a portion,

about 80 per cent, of the lower one on the right because it was pear shaped, and I wanted to be sure that it did not contain a minute adenoma. I left the top of it, being quite sure that there was no adenoma in this remnant. About 20 per cent of each of the other three glands was resected, to see if Dr. Castleman could find any evidence of secondary hyperplasia.

Clinical Diagnoses

Parathyroid adenoma.
Nephrolithiasis.

Dr. Aub's Diagnosis

Renal stones due to pyelonephritis and secondary renal calcification.

Anatomical Diagnoses

(**Nephrolithiasis.**)
Normal parathyroid glands.

Pathological Discussion

Dr. Castleman: We sectioned all four glands and found them to be normal. In each, more than half the gland was composed of fat cells and the epithelial cells seemed to be normal in size. If this had been secondary hyperplasia or an adenoma, the fat cells would have been replaced in the case of hyperplasia by an increase in the number of cells and in the case of adenoma by an increase both in the number and in the size of the cells, in other words, hyperplasia and hypertrophy. There was no suggestion of any degree of hyperplasia or hypertrophy.

Dr. Aub: Feeling much more confident, I should like to add that this work of Randall's is extremely interesting. The condition that he has described occurs in youth as well as in later life. The papillae of the kidneys develop small areas of calcification below the mucous membrane, which eventually ruptures. With the passage of urine over this denuded calcification the stage is set for the accumulation of a stone of any kind. This must be a source of a fair number of stones.

Dr. Castleman: Has there been any change in the patient's condition since operation?

Dr. Cope: The patient was given penicillin before and after operation to decrease the urinary infection, and he told me that after that for the first time in several years he was free of discomfort in the back bilaterally, suggesting that the infection had diminished. That is the only thing that he noticed clinically.

Dr. Albright: A culture of the urine gave no growth.

CASE 32022

Presentation of Case

A fifty-one-year-old electrician entered the hospital complaining of chills and fever.

Eighteen months before admission, the patient drank some pasteurized but apparently spoiled milk. A short time later he noticed chills, fever and sweating. These symptoms occurred daily and seemed worse in the middle of the day. There were occasional afebrile days, but they became less frequent. During the next two months he lost 10 pounds and became extremely weak, with a sensation of fullness in the left upper quadrant of the abdomen. He was bedridden for ten weeks. A blood culture was said to be "positive" for *Brucella abortus*. A blood transfusion resulted in marked improvement. He returned to work and felt well during the next seven months. At the end of that time he noticed enlargement of the cervical lymph nodes. He again complained of chills and fever and was hospitalized. He received a transfusion from a patient who had recovered from undulant fever and showed great improvement. Two months later he again became ill and entered a local hospital. At that time an eruption of red spots that lasted a day appeared over the entire body. A blood transfusion again improved his condition, but two months later his symptoms recurred. One month before admission he entered another hospital with chills, fever, sweating and a sensation of fullness in the left upper quadrant. At that time transfusions resulted in no improvement. He was treated with sulfadiazine. Anemia and leukopenia, which had been present since the onset of the illness, persisted. Blood cultures and agglutination tests were negative for *Br. abortus*. The blood was negative for malarial parasites.

The past history was negative.

Physical examination showed a poorly nourished, pale patient who was perspiring profusely. The mucous membranes were pale, and the tongue was smooth. Numerous fused, firm, nontender immovable nodes were palpable on the right side of the neck, and many small, firm, discrete nodes were present in the left cervical region. The heart and lungs were normal. The spleen was firm and extended to the level of the umbilicus. The liver was percussed one fingerbreadth below the right costal margin on deep inspiration.

The temperature was 97.6°F., the pulse 80, and the respirations 20. The blood pressure was 110 systolic, 80 diastolic.

The red-cell count was 3,360,000, with 10.5 gm. of hemoglobin. The white-cell count was 2600, with 63 per cent neutrophils and 28 lymphocytes. Platelets were seen in normal numbers. The urine was red and acid and had a specific gravity of 1.022. It gave a + test for albumin, and the sediment contained hyaline casts as well as 2 to 5 white cells and an occasional red cell per high-power field. The nonprotein nitrogen was 25 mg. per 100 cc., and the total serum protein 4.1 gm., with 2.8 gm. of albumin and 1.3 gm. of globulin. The serum chloride was 97 milliequiv. per liter. Tests for agglutinins against typhoid and brucellar organisms were negative. A culture of the urine showed a moderate number of

colonies of *Staphylococcus aureus* and rare diphtheroid colonies. Stool culture was negative for pathogens.

An x-ray film of the chest showed large clear lung fields. The diaphragm was somewhat low in position. The heart and upper mediastinum were not remarkable.

A lymph-node biopsy was performed.

Differential Diagnosis

Dr. Lowrey Davenport: We have a man of fifty-one years whose essential symptom during the period of eighteen months was recurrent fever and whose significant positive findings on physical examination were enlargement of the cervical lymph nodes, enlargement of the spleen and moderate enlargement of the liver.

In the differential diagnosis we have to consider all the possibilities that would cause a relapsing fever over a period of eighteen months, considering separately, if you will, all the possibilities causing cervical adenopathy and enlargement of the spleen, to see if we can tie in the physical findings and the symptomatology. We are faced at the onset with the gratuitous finding given in the early part of the history of a positive culture for *Br. abortus*. Subsequent cultures, however, were negative, as were agglutination tests for typhoid fever and brucellosis. We have to decide what we are going to do about this one positive culture. It seems unlikely in a case that has been observed for a period of eighteen months with repeated subsequent negative cultures and agglutination tests that the finding of a single positive culture for *Br. abortus* should be accepted. We are told that the patient did drink spoiled milk, but we are also told that it was pasteurized, and it is inconceivable that *Br. abortus* could have been present in the milk after pasteurization. Furthermore, his clinical record over a period of eighteen months is decidedly against undulant fever: he developed a rather marked anemia, a cervical adenitis and an enlarged firm spleen, all of which are unusual in brucellosis. The course of undulant fever over a period of eighteen months should have given a better clue to the diagnosis, and in spite of the history of his having drunk spoiled milk and the one positive culture, I shall throw out that diagnosis.

In considering fevers of unknown origin lasting for this period of time, we have the more frequent types, such as hidden tuberculosis or a hidden focus of pyogenic infection, and several obscure types. Recurrent malaria appears to have been ruled out by negative smears. I believe that recurrent rheumatic fever can also be ruled out: the patient was carefully observed for eighteen months without evidence of joint involvement. When one is faced with a situation of this sort, with a hidden focus of infection, the liver is usually the cause of the prolonged fever. We are all aware that the ordinary types of pyogenic infection of long duration show intermittent leukocytosis, and certainly after one and a half years they should give definite information concerning the site and location of the focus of sepsis. We can rule out pulmonary tuberculosis since the patient had a completely clear x-ray film, at admission, but lymph-node tuberculosis must be considered in a separate category.

In a long continued fever of this sort, with progressive anemia, we have to think of the possibility of subacute bacterial endocarditis. There was no evidence, however, of peripheral emboli; also, the blood showed a marked leukopenia rather than a leukocytosis. Furthermore, the heart was normal on physical examination. Thus, at least three of the several criteria for making the diagnosis of subacute bacterial endocarditis are lacking.

There are several rare types of prolonged fever that should be mentioned in passing. Almost by the definition of his symptoms, this man had relapsing fever. Could this have been the type of relapsing fever that is due to spirochetal organisms? I think that we can exclude this diagnosis on several grounds. It has been reported, either louse borne or tick borne, in North America, but it is an exceedingly rare condition in this part of the world. Also, it is usually, in fact almost invariably, associated with leukocytosis, and we should not expect to have the finding of enlarged cervical nodes or an enlarged spleen. So most of the common infectious types of fever can be thrown out of the picture.

We should now consider the finding of large nontender immovable cervical nodes, with an enlarged spleen, which was firm, and a moderately enlarged liver. In considering a diagnosis that ties up the enlarged cervical nodes and the enlarged spleen, two things immediately come to mind. The first and likeliest possibility on that basis is Hodgkin's disease. We are all familiar with the particular form of Hodgkin's disease that goes under the name of "Pel-Ebstein type," which is characterized by recurrent episodes of septic temperature over a period of months or years; there are lymph-node enlargement and progressive anemia, and the disease finally leads to death. This is an intriguing possibility and should be seriously considered in arriving at a presumptive diagnosis. Since we are faced with confusing laboratory data and a confusing clinical picture for one and a half years, we must take it for granted that the diagnosis that we shall make before hearing the report of the biopsy is bound to be presumptive. What is against a diagnosis of Hodgkin's disease? This man, who had been hospitalized several times in a year and a half, with repeated blood examinations, had never shown any of the characteristic changes of Hodgkin's disease in the blood picture. Although the blood findings in Hodgkin's disease are never pathognomonic, a relative leukocytosis is sometimes observed during the course of the disease and an eosinophilia is often present. We have no record here of an early

blood examination, but the meager findings we do have regarding the later blood picture seem to cast some doubt on this being the usual type of Hodgkin's lymphoma.

Could these have been tuberculous cervical lymph nodes? It is possible for such nodes to cause long continued intermittent fever, although such a condition now is rare, owing to the widespread pasteurization of milk. A septic type of fever, however, accompanied by profuse sweating, and other evidence of sepsis, as described in this individual, are rare unless the nodes are involved by secondary infection. We are told that the nodes were nontender and that they showed no evidence of suppuration. So we can assume that a noncomplicated tuberculous adenitis did not cause this picture.

What other forms of blood dyscrasia can cause enlarged cervical nodes and a large spleen? This man was repeatedly observed, but apparently showed no evidence of leukemia in the peripheral blood. One must always consider the possibility of leukemia in an aleukemic phase, better termed a "subleukemic" phase, since the leukemic elements are present if searched for. It seems likely that sometime during the course of eighteen months the typical picture of leukemia in the peripheral blood would have been found, so that the blood dyscrasias, particularly one of the leukemias, can be discarded.

We are faced with another intriguing possibility in a patient of this sort, that is, the disseminated form of lupus erythematosus. It is a condition that has come to be recognized more and more as an entity that should be considered in the differential diagnosis of long continued obscure fevers and obscure conditions with protean manifestations. Undoubtedly, the butterfly distribution of the erythema and the erythema itself have been greatly overemphasized. We now recognize that it is possible to have hidden forms of disseminated lupus without the typical erythematous eruption and the butterfly distribution. Lupus erythematosus can cause enlargement of the cervical nodes and an enlarged spleen. Over a period of eighteen months it can produce a recurrent intermittent type of fever and a progressive anemia, and also a change in the blood picture, which in the late stages is almost invariably a leukopenia. There are other things in the history that should make us consider this diagnosis carefully. There is no report of the chronic skin change that frequently accompanies the disease, but on one hospital admission the patient had a transient erythematous rash. In the late stages of disseminated lupus, because it is a disease of protean manifestations with generalized disorder of various organs in the body, one quite frequently obtains evidence of involvement of the kidneys, such as red cells, albumin and casts, which were found in this man's urine.

How much emphasis should we lay on the reports of the urine cultures? If this man had had a focus of infection in the kidneys sufficient to give this type of clinical course over one and a half years, I should think that the early symptoms would have been more prominent than those given in the history.

We are thus finally faced with two possibilities—Hodgkin's disease, with the Pel-Ebstein type of temperature, and the disseminated form of lupus erythematosus. We must now decide which better fits the picture. One thing is definitely against disseminated lupus. It is a disease with a predilection for young women, in certain series the incidence having been as high as 90 per cent. That does not militate completely against the diagnosis, however, because the disease has been reported in men as old as seventy. Although the chances are against lupus because of its usual sex distribution, it must be seriously considered because of the clinical course. There are two or three things against Hodgkin's disease. In the first place, the cervical nodes are described as being fixed, whereas the nodes in Hodgkin's disease, as differentiated from tuberculous nodes, are usually said to be freely movable. Also, as previously mentioned, there is no indication that during the course of eighteen months he had any of the specific changes in the blood picture that frequently accompany Hodgkin's lymphoma. The age and sex distribution, however, favor this diagnosis. It is of common occurrence in men in the third and fourth decades, whereas disseminated lupus usually occurs in the second and third decades in women. Realizing that any diagnosis is only presumptive and that a positive diagnosis can be given only by the microscopical findings, I shall put down my first choice as disseminated lupus, with Hodgkin's disease second.

DR. WYMAN RICHARDSON: This patient came in while I was on service. One interesting feature was that the temperature chart showed a double peak. I do not know what that means. I might add that the description of the nodes in the record is inaccurate; they formed a real mass, which I do not believe one sees in disseminated lupus. In the cases that I have seen the enlargement has been more diffuse and not so localized. I should think that that was against disseminated lupus. I also considered the possibility of sarcoid, but I had never seen such a severe case with as high a fever. I, too, considered malaria, but the smears were negative. So I came down to a diagnosis of Hodgkin's disease, with a possibility of sarcoid.

So far as the blood picture of Hodgkin's is concerned I do not believe that one can lean on it from the negative point of view; sometimes there are suggestive features from a positive point of view. The other point I should like to make is that most patients with a big spleen have a leukopenia whatever the cause, excluding, of course, true leukemia, polycythemia and infarction of the spleen.

Clinical Diagnosis

Hodgkin's disease?
Sarcoid?

Dr. Davenport's Diagnosis

Disseminated lupus erythematosus?
Hodgkin's disease?

Anatomical Diagnosis

Malignant lymphoma, Hodgkin's sarcoma type, involving cervical and retroperitoneal lymph nodes, spleen and bone marrow.

Pathological Discussion

Dr. Benjamin Castleman: The biopsy showed Hodgkin's disease of the sarcoma type, a very malignant form, with many Sternberg–Reed cells. The patient received x-ray treatment but died a few weeks later. At autopsy the spleen was tremendous in size, weighing over 1100 gm., and was infiltrated with yellowish nodules. Microscopically it was replaced with Hodgkin's sarcoma. There is a difference of opinion whether Hodgkin's disease is a granuloma or a neoplasm. In this laboratory we believe that it is a neoplasm rather than a granuloma, and a case like this seems to confirm our belief.

The finding of *Br. abortus* in a case of Hodgkin's disease brings up the recent work of Forbus and his colleagues.* They were able to find Brucella in a large percentage of cases of Hodgkin's disease. They also injected the organisms into animals and produced a picture somewhat similar to, but certainly not characteristic of, Hodgkin's disease. This work has not been confirmed, and I believe that most pathologists are skeptical of this connection.

*Forbus, W. D. et al. Studies on Hodgkin's disease and its relation to infection by Brucella. *Am. J. Path.* 18:745-748, 1942.

The abdominal and cervical nodes and the bone marrow were diffusely involved. The bone-marrow involvement might have accounted for the anemia.

Dr. Richardson: It was not the blood picture of myelophthisic anemia.

Dr. Castleman: Nevertheless, the vertebral marrow was almost completely replaced by sarcoma.

Dr. Chester M. Jones: I should like to comment on the question of brucellosis. I saw a good many of Forbus's cases and talked with the group at Duke University only a few weeks ago. I think that they are in general agreement now that brucellosis has nothing to do with Hodgkin's disease. In the neighborhood of Durham brucellosis is endemic and a good many people in the community have it. Its presence in Hodgkin's disease was simply an incidental finding.

Dr. Castleman: Most people have thought that right along.

Dr. J. H. Means: Do we see Hodgkin's disease with tuberculosis any more?

Dr. Richardson: We do not see it nearly so much as formerly.

Dr. Castleman: In the last ten or fifteen years we have seen only two or three cases of Hodgkin's disease associated with tuberculosis.

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NEW YORK CITY RESTRICTS THE SALE OF PENICILLIN

WHENEVER a new agent becomes available and is proved useful in the treatment of disease, a problem arises concerning the extent to which its sale and distribution should be controlled. In general, federal and local authorities have been loath to apply restrictions, and when they have made attempts to do so, they have not always succeeded in withstanding the pressure from pharmaceutical and other manufacturers who are anxious to profit from the new developments. Unfortunately, many manufacturers are always ready to cash in on any wave of popularity and are not always interested in whether or not the best public good is served by the widespread use of the product from which they derive their profits. On the other hand, the more reputable pharmaceutical firms have learned that in the long run their own best interests coincide with those of the public welfare and are best served by the carefully controlled production and distribution of products the value of which is thoroughly proved.

There are now available federal laws and regulations governing the sale and distribution of many medicinal products. In general, the purpose of these laws is to prevent the sales in interstate commerce of new substances that may be harmful and to permit the sale of such medicinal agents only after they have been proved to be of value. The Food and Drug Administration has also attempted to control advertising and labeling so as to prevent the making of unwarranted claims, while at the same time permitting the dissemination of information concerning the proper uses of new remedies.

Since most of the known drugs and biologic agents that are therapeutically active may also produce harmful side-effects, the restrictions on the sales of such agents are readily justified. The sulfonamide drugs can readily be included in this class, and there is little doubt that serious harm may result from their uncontrolled use. Regulations forbidding over-the-counter sales of sulfonamides are readily justified on this score. The demonstration of serious sensitization from the use of these agents in ointments, bandages, cosmetics, shaving creams and similar products is sufficient reason for restricting their sales in this manner. The physician who prescribes these products implicitly assumes the responsibility for seeing that they are used in a manner that minimizes their dangerous side-effects.

In the case of penicillin, toxic reactions, although by no means absent, are relatively infrequent and usually not severe. Many manufacturers are undoubtedly most anxious to have the opportunity of including the word "penicillin" on the labels of their products and to use this as a selling point, irrespective of the amount, activity or usefulness of the material that is incorporated. Great pressure will probably be brought to bear, as soon as the antibiotic is produced at a sufficiently low cost, to obtain permission to include penicillin in oint-

ments, bandages, eye lotions, shaving creams and many other products in which the particular manufacturers are interested. That such practices are not in the public interest is readily apparent to physicians, but it remains to be seen whether it is possible legally to prevent them. The facts that the manufacture of penicillin is largely in the hands of large and reputable manufacturers and that each batch must be carefully tested and approved by the Food and Drug Administration, may serve as a deterrent. Unscrupulous manufacturers, however, are not likely to submit to such rigid control, since the increased cost would make their ventures less profitable.

The Department of Health of the City of New York* has recently added a section to its Sanitary Code that restricts the sale of preparations containing penicillin or other antibiotic drugs to persons presenting a written prescription of a physician, dentist, podiatrist or veterinarian, except in the case of preparations that the Department of Health may specifically exempt from the provisions of that section. This action was based on the fact that indiscriminate use of penicillin and other antibiotics may be dangerous. One of the hazards listed is that undertreatment in many diseases may alleviate the symptoms of illness without curing the disease, which, in turn, may continue to produce damage in the patient and to be communicable to other persons. Other reasons given were that "there is accumulating evidence of the possible importance of the development of penicillin-fast strains in persons who are inadequately treated" and that "antibiotic drugs have been so recently developed that there has not been sufficient time to determine the possible toxic reactions." Whenever evidence is presented to the Department of Health that a certain preparation containing an antibiotic is entirely harmless yet effective in the treatment of disease, the matter of exemptions is duly considered. Under this provision an exception has already been made that permits the over-the-counter sale of bandages that contain a specified concentration of tyrothricin for external application to the human skin.

The action of the health authorities of New York City is certainly a step in the right direction. The principles on which this step was based are particularly commendable. It is doubtful that many other local authorities would have been willing to apply those principles to the dispensing of penicillin; on the other hand it is hoped that this regulation will lead to similar steps elsewhere.

*Sale of antibiotic drugs restricted. *Quart. Bull. Dept. of Health, City of New York* 13:33, 1945.

MASSACHUSETTS MEDICAL SOCIETY POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

ROUTINE BLOOD TESTS FOR SYPHILIS

THE value of routine blood tests for syphilis in large groups of people has been demonstrated again and again as an essential item in control of the disease. If further evidence is needed, a recent analysis of 5000 inductees in whom positive serologic tests for or a definite history of syphilis were found is convincing.†

In nearly 60 per cent of the group syphilis had not been recognized and consequently had not been previously treated. At the same time it is pointed out that more evidence than a single positive serologic test is necessary to establish a diagnosis of syphilis. Thus, in 11 per cent of the cases no diagnosis of syphilis could be made after a careful history, detailed physical examination, spinal-fluid examination and further blood examinations. In another 11 per cent the syphilis had apparently

†Zellermayer, J. Syphilis in inductees: analysis of 5000 cases. *J. Ven. Dis. Inform.* 25:194-198, 1945.

been adequately treated, since no evidence of the disease was found. In 13 per cent, the disease had progressed to a point where cerebrospinal, cardiovascular or other complications were demonstrable. In the remainder (65 per cent) the disease had been inadequately treated or untreated, and although evidence of disease still persisted, there were no complications. The last group contained a few cases of primary and secondary syphilis, but the bulk of them were early latent cases, with a few late latent cases; in other words, the disease was recognized before any of the serious organic changes had appeared.

The report emphasizes the need of careful diagnostic work to avoid the treatment of nonsyphilitic persons. The positive serologic test is a pointer toward the diagnosis of syphilis, but too much reliance should not be placed on the result of a single test and the patient's history should be obtained and his examination should be conducted with proper care.

MASSACHUSETTS MEDICAL SOCIETY

BLUE TRIANGLE EXPANDS

Since the inauguration of the Blue Triangle late in 1944 much progress has resulted in making this plan available on both a statewide and a nationwide basis. The Massachusetts Medical Society, the Massachusetts Dental Society and the Massachusetts Bankers Association have co-operatively produced and promoted the Blue Triangle to the extent that this service is now available in forty-five Massachusetts banks. In one bank alone it is reported that more than sixty of their local physicians and dentists are now using this modern method to assist their patients in paying for essential medical and dental care.

Inquiries about the Blue Triangle have been received from various sources in forty-five of the forty-eight states. This is most encouraging, for once again Massachusetts has led the way with a splendid example of co-operation directed to the public welfare.

The medical profession has already developed the Blue Cross — a prepayment plan for hospitalization — and the Blue Shield — a prepayment plan for medical care. Both plans are like insurance — fine if there is a loss and if the particular loss is fully covered. But certain people are not so insured, or their particular expenses are not covered. The Blue Triangle fills this need.

For the benefit of those doctors who have just returned from the service and may not be acquainted with the Blue Triangle, and for those who may have overlooked investigating its possibilities, the following is a brief outline of how the plan operates. When the patient contracts for or has accrued a bill larger than he can ordinarily pay out of a week's or month's wages, he is asked by the physician if he would prefer to pay the sum due in regular installments over a six-month to twelve-month period at the local bank that is participating in the plan. If the patient agrees, he signs a note, which the doctor transfers to the bank. The patient then repays the bank in monthly installments. In this way the doctor receives his money from the bank at the time he sends them the note, less a reserve (ordinarily 10 per cent). This reserve is paid to the doctor on final payment of the note by the patient. In this way the patient obtains a loan when he needs it with a minimum of effort and at a rate lower than he could obtain on a regular personal loan.

The Blue Triangle, although it has made great strides in the past year, is expected to be of even greater service in the years to come. With so many men and women coming out of the service who will again be calling on the family doctor, the Blue Triangle is bound to be of use to them when unexpected medical expenses arise. This is especially true when people are able to buy automobiles, appliances and the many other things they are waiting for. Furthermore, returning doctors who are reestablishing their practices will find it helpful from a financial standpoint.

The Blue Triangle is truly a health-financing service. Additional information regarding the plan may be obtained from the Massachusetts Medical Society.

DEATHS

DAVENPORT — Benita C. Davenport, M.D., of Middleton, died December 20. She was in her fifty-eighth year.

Dr. Davenport received her degree from the Boston University School of Medicine in 1930. She became assistant physician at Essex Sanatorium in 1933 and served as senior physician from 1937 until ill health forced her to retire in 1943. She was a fellow of the American Medical Association and a member of the American Tuberculosis Association, the American Sanatorium Association and the American Trudeau Society.

Two sisters survive.

DRUMMEY — Nicholas D. Drummey, M.D., of Dorchester, died December 18. He was in his eighty-first year.

Dr. Drummey received his degree from Harvard Medical School in 1887. He was the oldest alumnus of the Carney Hospital and was a consultant on the staff of the Faulkner Hospital. He was a fellow of the American Medical Association.

A son survives.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1945

RÉSUMÉ

Diseases	November 1945	November 1944	Seven-Year Median
Anterior poliomyelitis	50	35	7
Chancroid	1	2	*
Chicken pox	771	1025	1025
Diphtheria	18	28	21
Dog bite	644	590	592
Dysentery, bacillary	9	24	24
German measles	55	55	55
Gonorrhea	509	445	421
Granuloma inguinale	0	1	*
Lymphogranuloma venereum	1	2	*
Malaria	41	50	2
Measles	657	353	816
Meningitis, meningococcal	10	28	12
Meningitis, Pfeiffer-bacillus	3	5	2
Meningitis, pneumococcal	3	2	3†
Meningitis, staphylococcal	0	1	0†
Meningitis, streptococcal	0	0	0†
Meningitis, other forms	4	3	2†
Meningitis, undetermined	2	4	4†
Mumps	523	914	399
Pneumonia, lobar	92	162	233
Salmonella infections	7	4	5
Scarlet fever	466	776	704
Syphilis	391	420	420
Tuberculosis, pulmonary	273	217	215
Tuberculosis, other forms	9	11	17
Typhoid fever	3	1	2
Undulant fever	5	4	5
Whooping cough	659	498	557

*Made reportable December, 1943.

†Four-year average.

Comment

Cases of lobar pneumonia were reported less frequently during November, October and September than at any time during these same months since 1918; they were about one third the seven-year median.

The number of cases of scarlet fever during November was substantially below the seven-year median and approximately 30 per cent less than that during November, 1944.

Whooping cough remains higher this year than for comparable periods last year.

Cases of anterior poliomyelitis dropped from 136 in October to 50 in November. The latter figure, however, represents the highest number of cases for November since 1935, when 58 cases were reported.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Becket, 1; Boston, 7; Brockton, 1; Brookline, 1; Chelsea, 1; Chicopee, 1; Everett, 1; Fitchburg, 5; Groton, 1; Haverhill, 1; Lowell, 1; Lynn, 2; Malden, 2; Marlboro, 1; Medford, 1; Orleans, 1; Palmer, 1; Pepperell, 1; Pittsfield, 1; Princeton, 1; Quincy, 3; Reading, 2; Saugus, 1; Somerville, 1; Walpole, 2; Wareham, 2; Watertown, 1; Weymouth, 4; Worcester, 2; total, 50.

Diphtheria was reported from: Adams, 1; Boston, 6; Gloucester, 4; Lowell, 1; Malden, 1; Melrose, 1; New Bedford, 1; Somerville, 2; Winthrop, 1; total, 18.

Dysentery, amebic, was reported from: Boston, 1; total, 1.

Dysentery, bacillary, was reported from: Boston, 2; Braintree, 1; Cambridge, 2; Camp Edwards, 1; Lowell, 2; Malden, 1; total, 9.

Encephalitis, infectious, was reported from: Lawrence, 1; Worcester, 1; total, 2.

Lymphocytic choriomeningitis was reported from: Quincy, 1; total, 1.

Malaria was reported from: Boston, 8; Brookline, 1; Camp Edwards, 5; Cambridge, 2; Cushing General Hospital, 2; Dedham, 2; Everett, 2; Gloucester, 1; Lunenburg, 1; Lynn, 1; Fort Devens, 8; Milton, 1; Regional Hospital (Waltham), 5; Springfield, 1; Wakefield, 1; total, 41.

Meningitis, meningococcal, was reported from: Auburn, 1; Boston, 3; Fall River, 1; Framingham, 1; Marblehead, 1; North Adams, 1; Quincy, 1; Topsfield, 1; total, 10.

Meningitis, Pfeiffer-bacillus, was reported from: Cambridge, 1; Mansfield, 1; Randolph, 1; total, 3.

Meningitis, pneumococcal, was reported from: Brookline, 1; Natick, 1; Swampscott, 1; total, 3.

Meningitis, other forms, was reported from: Boston, 3; Milton, 1; total, 4.

Meningitis, undetermined, was reported from: Haverhill, 1; Worcester, 1; total, 2.

Salmonella infections were reported from: Boston, 1; Billerica, 1; Brookline, 1; Lowell, 1; Malden, 2; Salem, 1; total, 7.

Septic sore throat was reported from: Boston, 1; Fall River, 1; Lynn, 1; Newbury, 1; Somerville, 1; total, 5.

Tetanus was reported from: Boston, 1; Framingham, 1; total, 2.

Trichinosis was reported from: Boston, 2; total, 2.

Typhoid fever was reported from: Boston, 1; Lawrence, 1; Waltham, 1; total, 3.

Undulant fever was reported from: Brookline, 1; Taunton, 2; Waltham, 1; Worcester, 1; total, 5.

MISCELLANY

DR. DENNY RETIRES

Dr. Francis P. Denny retired in November of this year as health officer of Brookline, after thirty-two years of unique service to that community. Born in 1869, Dr. Denny graduated from Harvard College in 1891 and from Harvard Medical School in 1895. He served an internship at the Massachusetts General Hospital, studied a year and a half in Berlin and Vienna and established himself in practice in Brookline in 1897.

Among his contributions to public health were the establishment of a bacteriologic laboratory in Brookline in 1899, — the first in Massachusetts outside Boston, — the establishment of a tuberculosis pavilion as part of the Brookline Contagious Hospital in 1914 and the establishment of a dental dispensary in 1915. He inaugurated immunization against diphtheria in Brookline in 1923.

For twenty-six years Dr. Denny was responsible for the quarterly publication of the *Brookline Health Bulletin*, a sheet that is delivered to every household in the town, printing regularly the results of milk examinations, the rating of restaurants and much other timely health advice. During his term of office Brookline was three times the winner of the health-conservation contest sponsored by the United States Chamber of Commerce.

Dr. Denny, in the course of a long and useful career, has been president of the Massachusetts Public Health Association, the Norfolk District Medical Society and the Brookline Anti-Tuberculosis Society and a member of the Executive Committee of the Massachusetts Tuberculosis League, now being its honorary vice-president.

He has been succeeded as health officer by Dr. Alfred L. Frechette, a graduate of the University of Vermont Medical School in 1934 and of the Harvard School of Public Health in 1939. Dr. Frechette was with the United States Public Health Service in North Africa in 1943 and 1944 and has been personal representative of Director Herbert H. Lehman, of the United Nations Relief and Rehabilitation Administration, in Ethiopia. He has been secretary of the New Hampshire State Board of Health for the past two years.

LIST OF POTENTIALLY DANGEROUS MATERIALS USED IN INDUSTRY

The following list of potentially dangerous materials used in industry has been recently prepared by the Division of Occupational Hygiene, Massachusetts Department of Labor and Industry. The italicized items are trade names.

Material	Composition
Acetylene tetrachloride	Tetrachlorethane
Agate	Chiefly free silica
Aqua ammonia	Ammonium hydroxide (ammonia water)
Aqua fortis	Nitric acid
Aqua regia	Nitric and hydrochloric acids
Arochlor	Chlorinated diphenyl
Banana oil	Amyl acetate
Barco spot remover	Benzol (25%) and carbon tetrachloride (75%)
Bentasol	Petroleum naphtha
Blacosolv	Trichlorethylene
Carrene	Methylene dichloride
Caustic soda	Sodium hydroxide
Cecolene	Trichlorethylene
Celite	Diatomaceous earth (silica)
Ceresan	Contains ethyl mercury phosphate
Chalcedony	Chiefly free silica
Chertz	Chiefly free silica
Chlorex	Dichlorethyl ether
Chlorox	Sodium hypochloride

Material	Composition
Chrome green	Contains lead chromate
Chrome yellow	Lead chromate
Chrystobalite	Free silica
Circosolv	Trichlorethylene
Coal gas	Carbon monoxide
Coal tar naphtha	Contains xylene
DDT	Dichlor diphenyl trichlorethane
Diatomaceous earth	Chiefly free silica
Ditto (duplicating ink)	Methyl alcohol (90%)
Dowclene	Carbon tetrachloride
Dowicide	Chlorophenols
Dykanol	Chlorinated diphenyl
Fibrocel	Free silica and asbestos
Filter aid	Free silica
Flake caustic	Sodium hydroxide
Flint	Free silica
Freon 12	Dichlor difluoromethane
Fusel oil	Mostly amyl alcohols
Granite	Free silica (33%)
Gunk	Emulsifying agent in kerosene, with cresol (3-4%)
Halowax	Chlorinated naphthalene
Handyflux	Contains potassium fluoride
Hexone	Methyl isobutyl ketone
Kieselguhr	Chiefly free silica
Larvex	Sodium fluorosilicate
Litharge	Lead oxide
Lye	Potassium or sodium hydroxide
Mersol	Denatured alcohol, ethyl acetate and gasoline
Methyl acetone	Methanol, methyl acetate and acetone
Muriatic acid	Hydrochloric acid
Nevsol	Benzol (60%)
Oil of vitriol	Sulfuric acid
Pear oil	Amyl acetate
Perchlorethylene	Tetrachlorethylene
Per-clene	Tetrachlorethylene
Permachlor	Trichlorethylene
Petrobenzol	Petroleum naphtha
Phillsolv	Either trichlorethylene or tetrachlorethylene
Prussic acid	Hydrogen cyanide
Pyranol	Contains chlorodiphenyl
Pyrene	Carbon tetrachloride
Quartz	Free silica
Quartzite	Free silica
Sand	Chiefly free silica
Santocel	Free silica
Silica gel	Free silica
Skellysolv	Petroleum naphtha
Soda ash	Sodium carbonate
Solox	Denatured alcohol, ethyl acetate and aviation gasoline
Solvesso	Toluol, xylol and naphtha
Sovasol 5	Naphtha
Sovasol 73	Petroleum naphtha (41%), benzol (4%) and toluene (55%)
Sovasol 74	Petroleum naphtha (15%) and xylol (85%)
Stoddard solvent	High-boiling naphtha
Strippet (paint remover)	May or may not contain benzol
Synasol	Denatured alcohol (formula 1), ethyl acetate and aviation gasoline
Taxite (paint remover)	Benzol (30%)
Tollac	Benzol (50%)
Triad	Trichlorethylene
Triasol	Trichlorethylene
Triclene	Trichlorethylene
Trielin	Trichlorethylene
Tripoli	Chiefly free silica
Troluoil	Petroleum naphtha
Tromex	Trichlorethylene
V M and P naphtha	Petroleum naphtha
Varsol	High-boiling naphtha
Wood alcohol	Methanol

BOOKS RECEIVED

The receipt of the following books is acknowledged and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Trauma in Internal Diseases: With consideration of experimental pathology and medicolegal aspects. By Rudolf A. Stern, M.D., assistant attending physician, City Hospital, New York City. With a foreword by Francis C. Wood, M.D., director of laboratories and of the Radiotherapy Department, St. Luke's Hospital, New York. 8°, cloth, 575 pp. New York: Grune and Stratton, 1945. $6.75.

The author has endeavored to present a series of critical case histories, especially those with exhaustive post-mortem findings, along with a description of experimental work, which has either confirmed or refuted the assumption of trauma as an etiologic factor for many internal diseases. In writing the text, the author has reviewed a great mass of literature represented in a bibliography of forty-three pages, which will be found at the end of the book. The material covers the whole range of internal diseases.

New Steps in Public Health: Twenty-second annual conference of the Milbank Memorial Fund, April 12-13, 1944. 8°, paper, 148 pp., with figures and tables. New York: Milbank Memorial Fund, 1945.

This second volume of the 1944 conference is devoted to a discussion of chronic disease, medical care and the role of emotional and social factors in illness and to a number of articles on the public-health aspects of nutrition, written by various persons.

On Modern Syphilotherapy, with Particular Reference to Salvarsan. By Albert Neisser, M.D. Translated by Isabelle von Sazenhofen Wartenberg. With a biography and bibliography by Frances T. Gardner. 4°, cloth, 42 pp., with frontispiece. Baltimore: Johns Hopkins Press, 1945. $1.00.

In this translation Miss Wartenberg has made available the paper of Dr. Niesser, first published in 1911. The text of the article is preceded by a short biography of the author, and appended to the text there is a complete bibliography of the writings of Dr. Neisser from 1874 to 1916. The volume is well bound and is a reprint from the *Bulletin of the History of Medicine.*

NOTICES

ANNOUNCEMENTS

Dr. James A. Halsted and Dr. Stanley Kimball announce the reopening of their offices with the Dedham Medical Associates at 743 High Street, Dedham. Dr. Curtis Prout will be associated with them.

Dr. Francis X. Walsh, having returned from active service with the United States Army, announces the reopening of his office at 632 Columbia Road, Dorchester.

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The regular meeting of the New England Society of Physical Medicine will be held at the Hotel Kenmore, Boston, on Wednesday, January 16, at 8 p.m. Dr. Arthur L. Watkins will speak on the subject "Studies in Electro-Diagnosis as Applied to Peripheral Nerve Injuries and Poliomyelitis."

A council meeting will be held at 6:00 p.m., followed by a dinner in the Empire Room at 6:30 p.m.

Members of the medical profession are cordially invited to attend the meeting.

PHI DELTA EPSILON SOCIETY

A meeting of the Phi Delta Epsilon Society will be held in the auditorium of Boston University School of Medicine, on Thursday, January 17, at 8:15 p.m. Dr. Samuel A. Levine will speak on the subject "Cardiac Murmurs."

Those interested are invited.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING

THURSDAY, JANUARY 17

FRIDAY, JANUARY 18

*9:00-10:00 a.m. Medical clinic. Isolation Amphitheater, Children's Hospital.
*9:00-10:00 a.m. War and Nerves. Major Merrill Moore, U.S.A. Joseph H. Pratt Diagnostic Hospital.
*10:00 a.m.-12:00 m. Medical staff rounds. Peter Bent Brigham Hospital.
10:50 a.m. Industrial Dermatosis. Dr. John G. Downing. (Postgraduate clinic in dermatology and syphilology.) Amphitheater, Dowling Building. Boston City Hospital.

MONDAY, JANUARY 21

*12:00 m.-1:00 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

TUESDAY, JANUARY 22

*9:00-10:00 a.m. Medical clinic. Infants' Hospital.
*12:15-1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

WEDNESDAY, JANUARY 23

*9:00-10:00 a.m. Surgical Aspects of Gastric and Duodenal Ulcer Dr. Edward A. Cooney. Joseph H. Pratt Diagnostic Hospital.
*12:00 m. Clinicopathological conference. Children's Hospital.
*7:15 p.m. Graduate seminar in pediatrics. Children's Medical Service, Amphitheater 3A, Massachusetts General Hospital.

*Open to the medical profession.

(*Notices continued on page xvii*)